U.S. DEPARTMENT OF COMMERCE
National Technical Information Service
PB-250 011

STATEMENT OF BASIS AND PURPOSE FOR THE NATIONAL INTERIM PRIMARY DRINKING WATER REGULATIONS

ENVIRONMENTAL PROTECTION AGENCY

DECEMBER 1975

063017

PB250011

STATEMENT of BASIS and PURPOSE
for the
National Interim Primary
Drinking Water Regulations

U. S. Environmental Protection Agency

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INFORMATION SERVICE
U S Department of Commerce
Springfield VA 22151

183

BIBLIOGRAPHIC DATA SHEET

1. Report No.

2.

3. Recipient's Accession No.

4. Title and Subtitle
Statement of Basis and Purpose for the National Interim Primary Drinking Water Regulations.

5. Report Date
December 1975

6.

7. Author(s)

8. Performing Organization Rept. No.

9. Performing Organization Name and Address
Environmental Protection Agency, Office of Water Supply (WH-550), Washington, D.C. 20460

10. Project/Task/Work Unit No.

11. Contract/Grant No.

12. Sponsoring Organization Name and Address

Environmental Protection Agency
Office of Water Supply
(WH-550), Washington, D.C. 20460

13. Type of Report & Period Covered

14.

15. Supplementary Notes

16. Abstracts

The Statement of Basis and Purpose for the National Interim Primary Drinking Water Regulations contains the concepts and rationale for arriving at the specific Maximum Contaminant Levels in the Regulations which were promulgated on December 24, 1975. In addition to the material in support of the Maximum Contaminant Levels for 10 inorganic chemicals, six organic chemicals, turbidity and microbiological contaminants, material is also included which provides the basis for the lack of maximum contaminant levels for certain other contaminants. Among the latter are sodium, sulfate, organics-carbon absorbable, cyanide, certain pesticides and general bacterial populations. Numerous literature citations are provided in support of the narrative material.

17. Key Words and Document Analysis. 17a. Descriptors

water contaminant
drinking water regulations
sodium
sulfate
organics-carbon absorbable
cyanide

17b. Identifiers/Open-Ended Terms

17c. COSATI Field/Group

PRICES SUBJECT TO CHANGE

18. Availability Statement

19. Security Class (This Report)
UNCLASSIFIED

20. Security Class (This Page)
UNCLASSIFIED

21. No. of Pages
183

22. Price
$7.50 2.25

APPENDIX

BACKGROUND USED IN DEVELOPING THE PROPOSED INTERIM PRIMARY DRINKING WATER REGULATIONS

The Proposed Interim Primary Drinking Water Regulations have been predicated on the best and latest information available at the time of their promulgation. The concepts and rationale included in this Appendix were used in arriving at specific limits and should enable those whose responsibility it is to interpret, apply, or enforce the Regulations to do so with understanding, judgment, and discretion.

A. SOURCE AND FACILITIES

B. MICROBIOLOGICAL QUALITY

C. CHEMICAL QUALITY

A - SOURCE AND FACILITIES

Mounting pollution problems indicate the need for increased attention to the quality of source waters. Abatement and control of pollution of sources will significantly aid in producing drinking water that will be in full compliance with the provisions of these Standards and will be esthetically acceptable to the consumer, but they will never eliminate the need for well designed water treatment facilities operated by competent personnel.

Production of water that poses no threat to the consumer's health depends on continuous protection. Because of human frailties associated with protection, priority should be given to selection of the purest source. Polluted sources should not be used unless other sources are economically unavailable, and then only when personnel, equipment, and operating procedures can be depended on to purify and otherwise continuously protect the drinking water supply.

Although ground waters obtained from aquifers beneath impervious strata, and not connected with fragmented or cavernous rock, have been considered sufficiently protected from bacterial contamination to preclude need for disinfection, this is frequently not true as ground waters are becoming polluted with increasing frequency, and the resulting hazards require special surveillance. An illustration of such pollution is the presence of pollutants originating either from sewage or industrial effluents.

Surface waters are subjected to increasing pollution and should never be used without being effectively disinfected. Because of the increasing hazards of pollution, the use of surface waters without coagulation and filtration must be accompanied by adequate past records and intensive surveillance of the quality of the raw water and the disinfected supply in order to assure constant protection. This surveillance should include a sanitary survey of the source and water handling, as well as biological examination of the supply.

The degree of treatment should be determined by the health hazards involved and the quality of the raw water. When in use, the source should be under continuous surveillance to assure adequacy of treatment in meeting the hazards of changing pollution conditions. Continuous, effective disinfection shall be considered the minimum treatment for any water supply except for ground waters in which total coliforms can be shown to be continually absent from the raw water. During times of unavoidable and excessive pollution of a source already in use, it may become necessary to provide extraordinary treatment (e.g., exceptionally strong disinfection, improved coagulation, and/or special operation). If the pollution cannot be removed satisfactorily by treatment, use of the source should be discontinued until the pollution has been reduced or eliminated.

The adequacy of protection by treatment should be judged, in part, on a record of the quality of water produced by the treatment plant and the relation of this quality to the requirements of these Regulations. Evaluation of adequacy of protection by treatment should also include frequent inspection of treatment works and their operation. Conscientious operation by well-trained, skillful, and competent operators is an essential part of protection by treatment. Operator competency is encouraged by a formal program leading to operator certification or licensing.

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1 See reference to relationship of chlorine residual and contact time required to kill viruses in section on Microbiological Quality.

Delivery of a safe water supply depends on adequate protection by natural means or by treatment, and protection of the water in the distribution system. Minimum protection should include programs that result in the provision of sufficient and safe materials and equipment to treat and distribute the water; disinfection of water mains, storage facilities, and other equipment after each installation, repair, or other modification that may have subjected them to possible contamination; prevention of health hazards, such as cross-connections or loss of pressure because of overdraft in excess of the system's capacity; and routine analysis of water samples and frequent survey of the water system to evaluate the adequacy of protection. The fact that the minimum number of samples are taken and analyzed and found to comply with specific quality requirements of these Standards, is not sufficient evidence that protection has been adequate. The protection procedures and physical facilities must be reviewed along with the results of water quality analyses to evaluate the adequacy of the supply's protection. Knowledge of physical defects or of the existence of other health hazards in the water supply system is evidence of a deficiency in protection of the water supply. Even though water quality analyses have indicated that the quality requirements have been met, the deficiencies must be corrected before the supply can be considered safe.

B - MICROBIOLOGICAL QUALITY

Coliform Group

Coliform bacteria traditionally have been the bacteriological tool used to measure the occurrence and intensity of fecal contamination in stream-pollution investigations for nearly 70 years. During this time, a mass of data has accumulated to permit a full evaluation of the sensitivity and specificity of this bacterial pollution indicator.

As defined in Standard Methods for the Examination of Water and Wastewater (1), "the coliform group includes all of the aerobic and facultative anaerobic, Gram-negative, non-spore-forming rod-shaped bacteria which ferment lactose with gas formation within 48 hours at 35° C." From this definition, it becomes immediately apparent that this bacterial grouping is somewhat artificial in that it embodies a heterogeneous collection of bacterial species having only a few broad characteristics in common. Yet, for practical applications to stream pollution studies, this grouping of selected bacterial species, which we shall term the "total coliform group," has proved to be a workable arrangement.

The total coliform group merits consideration as an indicator of pollution because these bacteria are always present in the normal intestinal tract of humans and other warm-blooded animals and are eliminated in large numbers in fecal wastes. Thus, the absence of total coliform bacteria is evidence of a bacteriologically safe water.

Some strains included in the total coliform group have a wide distribution in the environment but are not common in fecal material. Enterobacter aerogenes and Enterobacter cloacae are frequently found on various types of vegetation (2-5) and in materials used in joints and valves (6-7).

The intermediate-aerogenes-cloacae (I.A C) subgroups may be found in fecal discharges, but usually in smaller numbers than Escherichia coli that is characteristically the predominant coliform in warm-blooded animal intestines (8-10). Enterobacter aerogenes and intermediate types of organisms are commonly present in soil (11-14) and in waters polluted some time in the past. Another subgroup comprises plant pathogens (15) and other organisms of indefinite taxonomy whose sanitary significance is uncertain. All of these coliform subgroups may be found in sewage and in the polluted water environment.

Survival Times

Organisms of the I.A.C. group tend to survive longer in water than do fecal coliform organisms (16-18). The I.A.C. group also tends to be somewhat more resistant to chlorination then E. coli or the commonly occurring bacterial intestinal pathogens (19-22). Because of these and other reasons, the relative survival times of the coliform subgroups may be useful in distinguishing between recent and less recent pollution. In waters recently contaminated with sewage, it is expected that fecal coliform organisms will be present in numbers greater than those of the I.A.C. subgroup; but in waters that have been contaminated for a considerable length of time or have been insufficiently chlorinated, organisms of the I.A.C. subgroup may be more numerous than fecal coliform organisms (23).

Differentiation of Organisms

Because various numbers of the coliform group normally grow in diverse natural habitats, attempts have been made to differentiate the population in polluted waters, with specific interest directed toward those coliforms that are derived from warm-blooded animal contamination. In his pioneering research, MacConkey (23, 24) defined the aerogenes group in terms of certain fermentation characteristics, ability to produce indole, and reaction in the Voges-Proskauer test. Other developments refined techniques that progressed to differentiate the coliform group on the basis of indole production, methyl red, and Voges-Proskauer reactions, and citrate utilization (IMViC tests) into the *E. coli*, *Enterobacter aerogenes*, intermediate, and irregular subgroups (24-28).

In another approach to coliform differentiation, Hajna and Perry (29) and Vaughn, Levine, and Smith (30) further developed the Eijkman test (31) to distinguish organisms of fecal origin from those of nonfecal origin by elevating the incubation temperature for lactose fermentation. Geldreich, and associates, (31, 32) further refined the procedure and developed additional data to indicate the specific correlation of this elevated temperature procedure to the occurrence of fecal contamination.

Fecal Coliform Measurements

The fecal coliform bacteria, a subgroup of the total coliform population, does have a direct correlation with fecal contamination from warm-blooded animals. The principal biochemical characteristic used to identify fecal coliform is the ability to ferment lactose with gas production at 44.5 C. Research data have shown that 96.4 percent of the coliforms in human feces were positive by this test (10). Examination of the excrement from other warm-blooded animals, including livestock, poultry, cats, dogs, and rodents (33-34), indicate the fecal coliforms contribute 93.0 to 98.7 percent of the total coliform population. The predominant fecal coliform type most frequently found in the intestinal flora is *E. coli*. Occasionally, other coliform IMViC types may predominate for periods of several months before a shift occurs in type distribution. For this reason, it is more significant to be able to measure all coliforms common to the intestinal tract. In man, particularly, there is a significantly greater positive correlation with the broader fecal coliform concept (96.4 percent) than with identification of *E. coli* by the traditional IMVic biochemical reactions (87.2 percent).

Application to Treated Water

The presence of any type of coliform organism in treated water suggests either inadequate treatment or contamination after post-chlorination (23). It is true there are some differences between various coliform strains with regard to natural survival and their chlorination resistance, but these are minor biological variations that are more clearly demonstrated in the laboratory than in the water treatment system. The presence of any coliform bacteria, fecal or nonfecal, in treated water should not be tolerated.

Insofar as bacterial pathogens are concerned, the coliform group is considered a reliable indicator of the adequacy of treatment. As an indicator of pollution in drinking water supply systems, and indirectly as an indicator of protection provided, the coliform group is preferred to fecal coliform organisms. Whether these considerations can be extended to include rickettsial and viral organisms has not been definitely determined.

Sample Size

The minimum official sample volume cited in the earlier editions of the Drinking Water Standards and Standard Methods for the Examination of Water and Wastewater was either stated or implied to be 50 ml because of the requirement to inoculate a series of 5 lactose broth fermentation tubes, each with a 10 ml or 100 ml portion of the sample. Few laboratories ever routinely employed the larger portions in the multiple tube procedure because of the attendant problems of preparing, handling and incubating the larger sized sample bottles that are required. Thus, when the multiple tube procedure was used, it became a practice to examine only 50 ml. With the development of the membrane filter procedure for routine potable water testing, the examination of larger sample volumes became practical, limited only by the turbidity of water and excessive bacterial populations.

Since many water supplies are sampled infrequently during the month, it is statistically more meaningful to examine a large sample for greater test precision with reduced risk of failing to detect some low level occurrence of coliforms. Increasing the sample portion examined will tighten the base line sensitivity and is particularly important for measuring the coliform reduction capacity of disinfection that approaches the magnitude essential for control of waterborne virus. Mack et al (35) reported poliovirus type II could be isolated from a restaurant well water supply using a flocculant in the 2.5 gallon samples prior to centrifugation to concentrate the low density virus particles. Bacteriological examinations of 50 ml portions of the unconcentrated water samples were negative for coliforms. However, coliforms were found in the concentrated sediment pellets. Future studies on coliform to virus occurrence in potable water may require further tightening of the coliform standard, possibly to a one-liter base (36).

The recommendations to increase the sample size to 100 ml for bacteriological examinations of water is supported in the 13th Edition of Standard Methods where the larger volume is stated as preferred. A study of State Health Laboratory procedures indicates that 39 or 78 percent of these laboratory systems are currently using 4 oz sample bottles to collect 100 ml of sample, and 25 of these State Health Laboratory networks are examining all public water samples by the membrane filter procedure. These figures suggest that the stronger position now being proposed on a minimum sample size of 100 ml for statistically improved coliform monitoring is not unrealistic in terms of current practice.

Application to Source Waters & Untreated Potable Supplies

In the monitoring of source water quality, fecal coliform measurements are preferred, being specific for fecal contamination and not subject to wide-range density fluctuation of doubtful sanitary significance.

Although the total coliform group is the prime measurement of potable water quality, the use of a fecal coliform measurement in untreated potable supplies will yield valuable supplemental information. Any untreated potable supply that contains one or more fecal coliforms per 100 ml should receive immediate disinfection.

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Substitution of Residual Chlorine Measurement for Total Coliform Measurement

The best method of assuring the microbiological safety of drinking water is to maintain good clarity, provide adequate disinfection, including maintenance of a disinfectant residual, and to make frequent measurements of the total coliform density in the distributed water. In the 1962 U.S. Public Health Service Drinking Water Standards, the major emphasis was on the measurement of total coliform densities and a sampling frequency graph relating number of samples per month to population served was included. The sampling frequency ranged from two per month for populations of 2,000 and less to over 500 per month, for a population of 8 million.

The effectiveness of this approach for assuring microbiological safety was evaluated during the 1969 Community Water Supply Survey. The results of this evaluation by McCabe, et. al., (1) are paraphrased below.

Microbiological Quality

To determine the status of the bacteriological surveillance program in each of the 969 water supply systems investigated, records in the State and county health departments were examined for the number of bacteriological samples taken and their results during the previous 12 months of record. Based on this information, only 10 percent had bacteriological surveillance programs that met the "criteria," while 90 percent either did not collect sufficient samples, or collected samples that showed poor bacterial quality, or both. The table below summarizes the results.

Bacteriological Surveillance

Population	500 or Less	501 100,000	Greater than 100,000	All Populations
Number of Systems	446	501	22	969
	Percent of Systems			
Met Criteria	4	15	36	10
Did not meet Criteria	95	85	64	90

Sampling Frequency

Insufficient samples were taken in more than one of the previous 12 months of record from 827 systems (85 percent of the survey total). Even considering a sampling rate reduced by 50 percent of that called for in the criteria, 670 systems (69 percent of the survey total) still would not have collected sufficient samples.

Recommendation

The water utility should be responsible for water quality control, but the bacteriological surveillance collection requirements are not being met in most small water systems even though only two samples per month are required. A more practical technique must be developed if the public's health is to be protected. If all systems were chlorinated, a residual chlorine determination might be a more practical way of characterizing safety.

The validity of the recommendation that the measurement of chlorine residual might be a substitute for some total coliform measurements has been investigated by Buelow and Walton (2).

Because the recommended rate of sample collection could not be or were not being used, alternative methods of indicating safety were considered. One suggestion was to substitute the measurement of chlorine residual for some of the bacteriological samples. Since this method has the advantage of being easy to perform, and thus providing an immediate indication of safety. Further, data from London, U.K.; Cincinnati, Ohio; and the 1969 Community Water Supply Survey (CWSS) has shown that present sampling locations do not protect all consumers and that chlorine residual can be used to replace some coliform determinations.

Sampling Location

During 1965-66, the London Metropolitan Water Board using its Standards, made bacteriological examinations of 11,371 samples of water entering the distribution system, 947 samples taken from distribution reservoirs, 2,720 samples taken following pipeline breaks, and 689 samples from miscellaneous locations (complaints, hospitals, etc.). Most of the unsatisfactory results were associated with reservoir problems. Main breaks and miscellaneous samples were responsible for most of the remaining unsatisfactory samples.

Chlorine Residual

In Cincinnati during the 1969-70 period of free chlorine residual, approximately 24 samples were collected from each of 143 sampling stations. None of the samples from 116 of these stations showed presence of coliform, and 23 of the remaining sampling stations showed coliform bacteria in only one out of the approximately 24 samples examined.

At the other four stations where 2 or more coliform-positive tests were obtained from the 24 samples, three had no chlorine residual at the time the coliform-positive samples were collected. The question is raised, therefore, as to the need for examining samples routinely collected from a large number of stations scattered throughout the system without regard to the water's residual chlorine content. Maintaining a free chlorine residual of 0.2 mg/l in the Cincinnati, Ohio, distribution system reduced the percentage of coliform positives to about 1 percent. The table below from the CWSS data, shows that the presence of a trace or more of chlorine residual drastically reduced or eliminated total coliforms from distribution system samples.

Percent of Various Types of Water Supply Systems Found to Have Average Total Coliforms Greater than 1/100 ml

Type of System	Non-Chlorinated	Chlorinated No Residual	With Any Detectable Residual
Spring	39	17	0
Combined Spring and Well	41	28	0
Well	8	5	0
Surface	64	7	2
Combined Surface and Well	100	16	3

These findings indicate that *a major portion of a distribution system, exclusive of deadends, reservoirs, etc., could be monitored for bacteriological safety by the use of chlorine residual*. (Emphasis added.)

Therefore, when chlorine substitution is used, determination of total coliform densitities should be continued in problem areas, and some samples, as a check, should be collected in the main part of the distribution system.

These two studies led to the inclusion in the Regulations of Par. 141.21(h) on the substitution of chlorine residual tests for a portion of the required total coliform determinations. Par. 141.21(h) states that any substitution must be approved by the State on the basis of a sanitary survey. The following four items should be specified by the State.

1. The number and location of samples for which chlorine residuals are to be substituted.
2. The form and concentration of chlorine residual to be maintained;
3. The frequency of chlorine residual determinations; and
4. The analytical method to be used.

While each approval must be made individually, taking into account individual circumstances, the following may offer some guidance. The first requirement is the establishment of the relationship between chlorine residual and the absence of total coliforms in any given water. This may not be too difficult in larger supplies where both of these measurements are routinely made, but it might be quite difficult for the smaller purveyors (where the most help is needed) who have not been making either measurement.

The number and location of samples for which chlorine residuals are to be substituted

Total coliform measurements should continue to be made of the finished water as it enters the distribution system and at known trouble spots such as reservoirs and dead ends. Substitution can be considered in the free-flowing portion of the distribution system.

The chlorine residual to be maintained

In general, a low turbidity water with a free chlorine residual of about 0.2 mg/l at a pH of less than 8.5 will be free from total coliforms although these conditions may vary from water to water. However, a higher free chlorine residual or the use of some other disinfectant is required prior to the water entering the distribution system, where disinfection is practiced, if initial disinfection is to be adequate.

The frequency of chlorine residual determinations

Because the chlorine residual test is so easy to perform, it is reasonable to expect the substitution of several chlorine residual determinations for each total coliform test deleted. In this way wider coverage of the distribution system can be achieved, thereby increasing the protection to the consumer. Since, for maximum protection, chlorination must be continuous, it is also reasonable to expect that a minimum of one daily determination of chlorine residual be performed whenever the chlorine residual option has been chosen. By limiting the extent of substitution to 75% of the required bacteriological samples, a sufficient number of bacteriological samples will still be taken to enable the assessment of the adequacy of disinfection and to assure the continuity of water quality records.

The analytical method to be used

An analytical method free of interferences to eliminate false residuals must be recommended. For this reason the DPD method is specified.

Finally, when the chlorine residual option is in use and a free chlorine residual concentration less than that agreed to is measured at a sampling point, then a sample for total coliform analysis must be taken immediately from that point.

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General Bacterial Population

The microbial flora in potable water supplies is highly variable in numbers and kinds of organisms. Those bacterial groups most frequently encountered in potable waters of poor quality include: Pseudomonas, Flavobacterium, Achromobacter, Proteus, Klebsiella, Bacillus, Serratia, Corynebacterium, Spirillum, Clostridium, Arthrobacter, Gallionella, and Leptothrix (1-5). Substantial populations of some of these organisms occurring in potable water supplies may bring a new area of health risk to hospitals, clinics, nurseries, and rest homes (6-11). Although Pseudomonas organisms are generally considered to be non-pathogenic, they can become a serious "secondary pathogenic invader" in post-operation infections, burn cases, and intestinal-urinary tract infections of very young infants and the elderly population of a community. These organisms can persist and grow in water containing a minimal nutrient source of nitrogen and carbon. If Pseudomonas becomes established in localized sections of the distribution lines, it may persist for long periods and shed irregularly into the consumer's potable water supply (12). A continual maintenance of 0.3 to 0.6 mg/l free chlorine residual will suppress the development of an extensive microbial flora in all sections of the distribution network.

Flavobacterium strains can be prevalent in drinking water and on water taps and drinking-fountain bubbler-heads. A recent study of stored emergency water supplies indicated that 23 percent of the samples contained Flavobacterium organisms with densities ranging from 10 to 26,000 per 1 ml. *Flavobacterium* must be controlled in the hospital environment because it can become a primary pathogen in persons who have undergone surgery (13).

Klebsiella pneumoniae is another secondary invader that produces human infection of the respiratory system, genito-urinary system, nose and throat, and occasionally this organism has been reported as the cause of meningitis and septicema (14). *Klebsiella pneumoniae*, like *Enterobacter aerogenes*, (15) can multiply in very minimal nutrients that may be found in slime accumulations in distribution pipes, water taps, air chambers, and aerators.

Coliform Suppression

The inhibitory influence of various organisms in the bacterial flora of water may be important factor that could negate detection of the coliform group (16-17). Strains of *Pseudomonas*, *Sarcina*, *Micrococcus*, *Flavobacterium*, *Proteus*, *Bacillus*, *Actinomycetes*, and yeast have been shown to suppress the detection of the coliform indicator group (18-21). These organisms can coexist in water, but when introduced into lactose broth they multiply at a rapid rate, intensifying the factor of coliform inhibition (22). Suspensions of various antagonistic organisms in a density range of 10,000 to 20,000 per 1 ml, added to lactose tubes simultaneously with a suspension of 10 *E. coli* per 1 ml, resulted in reduction in coliform detection (19). This loss of test sensitivity ranged from 28 to 97 percent, depending on the combination of the mixed strains.

Data from the National Community Water Supply Survey (23) on bacteriological quality of distribution water from the 969 public water supplies were analyzed (Table 1) for bacterial plate count relationship to detection of total coliforms and fecal coliforms. It is interesting to note that there was a significant increase in total and fecal coliform detection when the bacterial counts increased up to 500 per 1 ml. However, further increase in the detection of either coliform parameter did not occur when the bacterial count per 1 ml was beyond 500 organisms. There was, in fact, progressively decreased detection of both coliform parameters as the bacterial count continued to rise. This could indicate an aftergrowth of bacteria in distribution system water or a breakpoint where coliform detection was desensitized by the occurrence of a large general bacterial population that included organisms known to suppress coliform recovery.

Control of the General Bacterial Population

Density limits for the general bacterial population must be related, in part, to a need to control undesirable water quality deterioration and practical attainment for water throughout the distribution system. This necessity for monitoring the general bacterial population is most essential in those supplies that do not maintain any chlorine residual in the distribution lines and in special applications involving desalinization. This bacteriological measurement would serve as a quality control on water treatment processes and sanitation of dis-

TABLE 1

BACTERIAL PLATE COUNT vs. COLIFORM DETECTION IN DISTRIBUTION WATER NETWORKS FOR 969 PUBLIC WATER SUPPLIES

General Bacterial Population*		Total Coliform		Fecal Coliform	
Density Range per 1 ml	Number of Samples	Occurrences	Percent	Occurrences	Percent
1 - 10	1013	47	4.6	22	2.2
11 - 30	371	28	7.5	12	3.2
31 - 100	396	72	18.2	28	7.1
101 - 300	272	48	17.6	20	7.4
301 - 500	120	30	25.0	11	9.2
501 - 1,000	110	21	19.1	9	8.2
1,000	164	31	18.9	5	3.0
TOTAL	2446	277	---	107	---

*Standard Plate Count (48 hrs. incubation, 35°C) tribution line sections and storage tanks that could be shedding various quantities of organisms into the system, thereby degrading the water quality.

Practical attainment of a low general bacterial population can best be judged by a study of data from the National Community Water Supply Survey. Data presented in Table 2 demonstrate the effectiveness of chlorine residual in controlling the general bacterial population in a variety of community water supply distribution systems. Although the number of samples on each distribution system in this special study was small, it does reflect bacterial quality conditions in numerous large and small water systems examined in each of the eight metropolitan areas and the entire State of Vermont.

These data indicate that the general bacterial population in distribution lines can be controlled to a value below 500 organisms per 1 ml by maintaining a residual chlorine level in the system. Increasing the chlorine residual above 0.3 mg/l to levels of 0.6 and 1.0 mg/l did not further reduce the bacterial population by any appreciable amount. Restricting such bacterial densities to a limit of 500 organisms per ml is, therefore, not only attainable in the distribution system, but is also desirable to prevent loss in coliform test sensitivity definitely observed at approximate densities of 1000 organisms per ml, thereby producing a safety factor of at least two.

TABLE 2

THE EFFECT OF VARYING LEVELS OF RESIDUAL CHLORINE ON THE TOTAL PLATE COUNT IN POTABLE WATER DISTRIBUTION SYSTEMS*

Standard Plate Count**	Residual Chlorine (mg/l)							
	0.0	0.01	0.1	0.2	0.3	0.4	0.5	0.6
< 1	8.1***	14.6	19.7	12.8	16.4	17.9	4.5	17.9
1 - 10	20.4	29.2	38.2	48.9	45.5	51.3	59.1	42.9
11 - 100	37.3	33.7	28.9	26.6	23.6	23.1	31.8	28.6
101 - 500	18.6	11.2	7.9	9.6	12.7	5.1	4.5	10.7
501 - 1000	5.6	6.7	1.3	2.1	1.8	0	0	0
>1000	10.0	4.5	3.9	0	0	2.6	0	0
Number of Samples	520	89	76	94	55	39	22	28

*Data from a survey of community water supply systems in 9 metropolitan areas (23)
**Standard Plate Count (48 hrs. incubation, 35°C)
***All values are percent of samples that had the indicated standard plate count.

Any application of a limit for the general bacterial population in potable water will require a definition of medium, incubation temperature, and incubation time so as to standardize the population to be measured. The 13th edition of Standard Methods for the Examination of Water and Wastewater does specify these requirements for a Standard Plate Count (SPC) to be used in collection of water quality control data. Because many organisms present in potable waters are attenuated, initial growth in plate count agar frequently is slow; thus, incubation time should be extended to 48 hours at 35 C. This time extension will permit a more meaningful standard count of the viable bacterial population. Samples must be collected in bottles previously sterilized within 30 days and adequately protected from dust accumulation. Examination for a Standard Plate Count should be initiated within 8 hours of collection. This time may be extended to periods up to 30 hours only if these samples are transported in iced containers.

With maintenance of a chlorine residual and turbidity of less than one Turbidity Unit, the need for a bacteriological measurement of the distribution system may become less critical. For this reason, it is recommended that such water supplies be monitored routinely for baseline data on the general bacterial population and correlated with chlorine residual and turbidity measurements in the distribution lines. It is also recommended that water plant personnel be alert to unusual circumstances that may make it desirable to monitor the general bacterial population more often in a check of water plant treatment efficiencies.

For these reasons, the general bacterial population should be limited to 500 organisms per 1 ml in distribution water. In theory, the limitation of the general bacterial population to some practical low level would also indirectly and proportionally limit any antagonistic organisms that could suppress coliform detection and reduce the exposure and dosage level for health effect organisms that might be present.

While no maximum contaminant level for general bacterial populations is included in the Interim Primary Drinking Water Regulations, it is recommended that the limit mentioned above be used as an operational guide in assessing the quality of drinking water delivered.

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these 3 water supplies were really adequately treated. Only one of the 8 poliomyelitis epidemics occurred in the United States, and this was the result of cross-connection contamination. Since Mosley's publication there have been three other reports of waterborne infectious hepatitis outbreaks in this country, all reportedly due to either sewage pollution of well water or cross-connection contamination. An estimated 20,000 - 40,000 cases of infectious hepatitis were reported in Delhi, India, in 1955-56 (2) attributable to a municipal water supply source heavily overloaded with raw sewage. This outbreak, however, was not accompanied by noticeable increases of typhoid fever or other enterobacterial diseases, suggesting that, in practice, the virus(es) of infectious hepatitis may be more resistant to chlorine or chloramines than are vegetative bacteria. Weibel and co-workers (3) listed 142 outbreaks of gastroenteritis during the period of 1946 to 1970 in which epidemiologic evidence suggested a waterborne nature. More than 18,000 persons were affected in these outbreaks. Mosley (1) suspected that a significant portion of these cases must have been caused by viruses.

It is well recognized that many raw water sources in this country are polluted with enteric viruses. Thus, water supplies from such sources depend entirely upon the treatment processes used to eliminate these pollutants. Even though the processes may be perfectly effective, an occasional breakdown in the plant or any marginal practice of treatment could still allow the pollutants to reach the finished water supplies. It should be noted that Coin and his associates (4) have reported the recovery of viruses from raw and finished waters in Paris, France. Coin estimated that the Paris water probably contained one tissue culture unit of virus per 250 liters. Very recently, Mack et al (5) reported that poliovirus was recovered in water from a deep well in Michigan. Although the well had a history of positive coliforms, coliforms and virus were not recovered from an unconcentrated water sample; only after a 2.5 gallon sample of water was subjected to high speed centrifugation were both virus and coliforms recovered. This study would seem to indicate that the present method of using the coliform test is not adequate to indicate the presence of viruses. In summary, in the United States, most waterborne virus disease outbreaks have resulted from contamination of poorly treated drinking water by sewage either directly or through cross-connections. Overt outbreaks of virus disease from properly treated municipal water supplies are not known to have occurred. Proper treatment of surface water usually means clarification followed by effective disinfection.

Chang (6), however, has theorized that some water supplies that practice only marginal treatment may contain low levels of human viruses, and that this small amount of virus might initiate infection or disease in susceptible individuals. He believes that such individuals might thus serve as "index cases" and further spread the virus by person-to-person contact. Whether this hypothesis is true, can be proved only by an intensive survey for viruses in numerous drinking water supplies in this country, and such a survey has never been conducted. If viruses were detected in a survey of drinking water supplies, it would be necessary to conduct in-depth epidemiological studies to determine if actual infection or disease was being caused by these agents. Additionally, it would be necessary to determine what modifications would be required in the water treatment processes to eliminate these viruses.

The relative number of viruses and coliform organisms in domestic sewage is important in assessing the significance of the coliform test and the "virus safety" of water. Calculations by Clarke et al (7) have indicated the following virus-coliform ratios in feces, sewage, and polluted waters.

Calculated Virus - Coliform Ratios

	Virus	Coliform	Ratio
Feces	200/gm	13×10^6/gm	1:65,000
Sewage	500/100 ml	46×10^6/100ml	1:92,000
Polluted Surface Water	1/100 ml	5×10^4/100 ml	1:50,000

It is apparent that coliform organisms far outnumber human enteric viruses in feces, sewage, and polluted surface water. It should be emphasized that these calculated ratios are only approximations and that they would be subject to wide variations and radical changes, particularly during a virus disease epidemic. Additionally, both bacteria and virus populations in sewage and polluted waters are subject to reductions, at different rates, from die-off, adsorption, sedimentation, dilution, and various other undetermined causes; thus, the coliform-virus ratio changes, depending upon conditions resulting from the combined effect of all factors present. Thus, one must take into consideration the most unfavorable conditions although they may be encountered very infrequently. Such conditions may impose considerable demands on the indicator system and treatment processes.

The efficacy of various water treatment processes in removing or inactivating viruses has recently been reviewed by Chang (6) and also in a Committee Report, "Engineering Evaluation of Virus Hazards in Water" (8). These reports indicate that natural "die-off" cannot be relied upon for the elimination of viruses in water. Laboratory pilot plant studies indicate that combination of coagulation and sand filtration is capable of reducing virus populations up to 99.7 percent if such treatments are properly carried out (9). It should be noted, however, that a floc breakthrough, sufficient to cause a turbidity of as little as 0.5 Turbidity Units, was usually accompanied by a virus breakthrough in a pilot plant unit seeded with high doses of virus (9). Disinfection, however, is the only reliable process by which water can be made free of virus. In the past, there have been numerous studies conducted on the chlorination of viruses. Recent work by Liu, et al (10), has confirmed early observations and has reemphasized two possible weaknesses in these early reports: (a) the number of virus types studied was very small, thus generalization on such results is not without pitfalls, (b) the early chlorination studies were usually conducted with reasonably pure virus suspensions derived from tissue cultures or animal tissue and may not represent the physical state of the virus as it exists under natural conditions (clumped, embedded in protective material, etc.) which would make the virus much more resistant to disinfectants. Thus, it is imperative that good clarification processes be used on turbid waters to reduce their turbidity levels that will ensure effective disinfection. Additionally, Liu's data show the wide variation in resistance to chlorine exhibited by viruses, e.g., four minutes were required to inactivate 99.99 percent of a reovirus population as contrasted to 60 minutes to achieve the same percent inactivation of coxsackievirus.

Virology techniques have not yet been perfected to a point where they can be used to routinely monitor water for viruses. Considerable progress on method development, however, has been made in the past decade. The methods potentially useful include: two-phase polymer separation (11), membrane filtration (12), adsorption on and elution from chemicals (13, 14, 15), and the gauze pad technique (16) to name a few. From the concerted efforts of virus-water laboratories throughout the world, it is hoped that a simple and effective method will become available for viral examination of water. In the interim, control laboratories having access to facilities for virus isolation and identification should be encouraged to use available procedures for evaluating the occurrence of human enteric viruses in treated waters.

As noted above, no simple and effective method for the viral examination of water is available at this time. When such a method is developed, and when there are sufficient data to provide the necessary basis, a maximum contaminant level for virus will be proposed.

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Turbidity

Drinking water should be low in turbidity prior to disinfection and at the consumer's tap for the following reasons:

(1) Several studies have demonstrated that the presence of particulate matter in water interferes with effective disinfection. Neefe, Baty, Reinhold, and Stokes (1) added from 40 to 50 ppm of feces containing the causative agent of infectious hepatitis to distilled water. They then treated this water by varying techniques and fed the resultant liquid to human volunteers. One portion of the water that was disinfected to a total chlorine residual after 30 minutes of 1.1 mg/l caused hepatitis in 2 of the 5 volunteers. A similar experiment in which the water was first coagulated and then filtered, prior to disinfection to the same concentration of total residual, produced no hepatitis in 5 volunteers. This was repeated with 7 additional volunteers, and again no infectious hepatitis occurred.

Chang, Woodward and Kabler (2) showed that nematode worms can ingest enteric bacterial pathogens as well as virus, and that the nematode-borne organisms are completely protected against chlorinations even when more than 90 percent of the carrier worms are immobilized.

Walton (3) analyzed data from three waterworks treating surface waters by chlorination only. Coliform bacteria were detected in the chlorinated water at only one waterworks, the one that treated a Great Lakes water that usually did not have turbidities greater than 10 turbidity units (TU), but occasionally contained turbidities as great as 100 TU.

Sanderson and Kelly (4) studied an impounded water supply receiving no treatment other than chlorination. The concentration of free chlorine residual in samples from household taps after a minimum of 30 minutes contact time varied from 0.1 to 0.5 mg/l and the total chlorine residual was between 0.7 and 1 mg/l. These samples consistently yielded confirmed coliform organisms. Turbidities in these samples varied from 4 to 84 TU, and microscopic examination showed iron rust and plankton to be present. They concluded "...coliform bacteria were imbedded in particles of turbidity and were probably never in contact with the active agent. Viruses, being smaller than bacteria, are much more likely to escape the action of chlorine in a natural water. Thus, it would be essential to treat water by coagulation and filtration to nearly zero turbidity if chlorination is to be effective as a viricidal process."

Hudson (5) reanalyzed the data of Walton, above, relating them to the hepatitis incidence for some of the cities that Walton studied plus a few others. A summary of his analysis is shown in Table I. Woodward does, however, in a companion discussion warn against over interpreting such limited data and urges more field and laboratory research to clearly demonstrate the facts.

TABLE I

FILTERED-WATER QUALITY AND HEPATITIS INCIDENCE, 1953

City	Average Turbidity TU	Final Chlorine Residual mg/1	Hepatitis cases/100,000 people
G	0.15	0.1	3.0
C	0.10	0.3	4.7
H	0.25	0.3	4.9
B	0.2	-	8.6
M	0.3	0.4	31.0
A	1.0	0.7	130.0

Tracy, Camarena, and Wing (6) noted that during 1963, in San Francisco, California, 33 percent of all the coliform samples showed 5 positive tubes, in spite of the presence of chlorine residual. During the period of greatest coliform persistence, the turbidity of this unfiltered supply was between 5 and 10 TU.

Finally, Robeck, Clarke, and Dostal (7) showed by laboratory demonstration that virus penetration through a granular filter was accompanied by a breakthrough of floc, as measured by an increase in effluent turbidity above 0.5 turbidity unit in a pilot unit seeded with an extremely high dose of virus.

These 7 studies show the importance of having a low turbidity water prior to disinfection and entrance into the distribution system.

(2) The 1969 Community Water Supply Survey (8) revealed that unpleasant tastes and odors were among the most common customer complaints. While organics and inorganics in finished water do cause tastes and odors, these problems are often aggravated by the reaction of chlorine with foreign substances. Maintenance of a low turbidity will permit distribution with less likelihood of increasing taste and odor problems.

(3) Regrowth of microorganisms in a distribution system is often stimulated if organic matter (food) is present. An example of this possibility occurred in a Pittsburgh hospital (9). One source of this food is biological forms such as algae which may contribute to gross turbidity. Therefore, the maintenance of low turbidity water will reduce the level of this microbial food and maintain a cleanliness that will help prevent regrowth of bacteria and the growth of other microorganisms.

(4) The purpose of maintaining a chlorine residual in a distribution system is to have a biocidal material present throughout the system so that the consumer will be protected if the integrity of the system is violated. Because the suspended material that causes turbidity may exert a chlorine demand, the maintenance of a low turbidity water throughout the distribution system will facilitate the provision of proper chlorine residual.

For these reasons, the limit for turbidity is one (1) Turbidity Unit (TU) as the water enters the distribution system. A properly operated water treatment plant employing coagulants and granular filtration should have no difficulty in consistently producing a finished water conforming to this limit.

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C - CHEMICAL QUALITY

The following pages present detailed data and the reasoning used in reaching the various limits.

In general, limits are based on the fact that the substances enumerated represent hazards to the health of man. In arriving at specific limits, the total environmental exposure of man to a stated specific toxicant has been considered. An attempt has been made to set lifetime limits at the lowest practical level in order to minimize the amount of a toxicant contributed by water, particularly when other sources such as milk, food, or air are known to represent the major exposure to man.

The Standards are regarded as a standard of quality that is generally attainable by good water quality control practices. Poor practice is an inherent health hazard. The policy has been to set limits that are not so low as to be impracticable nor so high as to encourage pollution of water.

No attempt has been made to prescribe specific limits for every toxic or undesirable contaminant that might enter a public water supply. While the need for continued attention to chemical contaminants of water is recognized, the Regulations are limited to need and available scientific data or implications on which judgments can be made. Standards for innumerable substances which are rarely found in water would require an impossible burden of analytical examination.

The following table indicates the percent of samples analyzed in the Community Water Supply Study which exceeded 75% of the 1962 PHS Drinking Water Standards limits. This table shows the relationship of the existing quality of water analyzed during the study to the drinking water standards in effect at that time.

PERCENT OF SAMPLES IN THE COMMUNITY WATER SUPPLY STUDY WITH VALUE EXCEEDING 75% OF EACH LIMIT IN THE 1962 DRINKING WATER STANDARDS

Constituent	DWS Limit	DWS Limit X 0.75	Percent of Samples Exceeding
Arsenic	0.05 mg/l	0.0375 mg/l	1.24%
Barium	1 mg/l	0.75 mg/l	0.08%
Cadmium	0.010 mg/l	0.0075 mg/l	1.45%
Chloride	250 mg/l	187.5 mg/l	1.56%
Chromium	0.05 mg/l	0.0375 mg/l	1.43%
Color	15 C.U.	11.25 C.U.	3.54%
Copper	1 mg/l	0.75 mg/l	2.47%
Cyanide	0.2 mg/l	0.15 mg/l	0.00%
Foaming Agents	0.5 mg/l	0.375 mg/l	0.08%
Iron	0.3 mg/l	0.225 mg/l	15.81%
Lead	0.05 mg/l	0.0375 mg/l	3.32%
Manganese	0.05 mg/l	0.0375 mg/l	11.91%
Nitrate	45 mg/l	33.75 mg/l	3.46%
Selenium	0.01 mg/l	0.0075 mg/l	8.35%
Silver	0.05 mg/l	0.0375 mg/l	0.00%
Sulfate	250 mg/l	187.5 mg/l	3.37%
Zinc	5 mg/l	3.75 mg/l	0.35%

DAILY FLUID INTAKE

For the purpose of these Regulations, a daily intake of water or water based fluids of two liters was assumed. This figure was taken as being representative of the fluid consumption of a normal adult male, and was obtained by consulting standard textbooks on physiology and numerous journal articles concerning water consumption.

It was realized that tremendous variation in individual consumption would exist, but since women and children drink less than the average man, it was decided that a large percent of the population would consume less than two liters a day.

There have been numerous reports of individuals or groups of persons who consume abnormally large quantities of water or water-based fluids. For example, the consumption of six liters of beer in a day (1, 2) is not unknown. However, it should be noted that anyone who consumes this quantity of beer would be getting more than 240 ml (1/2 pint) of pure alcohol which is close to the maximum tolerable dose for a day.

The Boy's Life Magazine (1971)(3) survey indicated that 8% of 10-17 year-old boys drink more than 8 soft drinks per day. This survey can be viewed from another angle and a statement made that 92% of such boys drink less than 8 soft drinks per day. It would probably be valid to state that the average consumption is far less than 8.

Guyton (1951)(4) properly indicates that diseased persons having diabetes insipidus consume great quantities of water a day but even raising the "daily fluid intake" to 6 liters a day would not protect these individuals who excrete up to 15 or more liters of urine per day. It might also be pointed out that diabetes insipidus is a relatively rare disease and that these patients could not be considered average consumers.

Welch, et al (5) show that at temperatures up to 75°F 2 liters or less of fluid are drunk per day by adult males.

Molnar, et al (6) found that average fluid intake in the desert was 5.90 liters per day with a standard deviation of $\pm$2.03 whereas average fluid intake in the tropics was 3.26 liters with a standard deviation of $\pm$1.09. These men were performing their normal duties including truck driving, guard duty, hiking, etc. Five percent of the men in the tropics drank as little as 1 liter a day.

Wyndham and Strydom (7) indicated that marathon runners lost between 1,500 and 4,200 ml of sweat in 20 miles of running at about 60°F. To replace their fluid that day would require from 2.5 to 5 liters of water.

In "Clinical Nutrition" (8) the normal water loss per day shown for a normal adult ranges from 1,500 ml - 2,100 ml. The breakdown for a 2,600 ml water intake is shown as 1,500 from fluids, 800 ml from food and 300 ml from metabolism.

In "Physiology of Man in the Desert"(9) the average intake of fluid for 91 men in the desert was 5.03 liters with a standard deviation of $\pm$1.67. This indicates that some men only drank three liters a day in a desert environment where temperatures went as high as 105°F.

In Best and Taylor's book, "The Physiological Basis of Medical Practice," (1945)(10) an average adult is shown to require 2,500 ml of water from all sources under ordinary circumstances. The sources of this water are shown as:

Solid and semisolid food	1200 cc
Oxidation of food	300 cc
Drinks (water, milk, coffee, beer, etc.)	1000 cc

This reference points out that cooked lean meat contains from 65 to 70 percent water.

It should be noted that certain references refer to water loss per day instead of drinking water intake. Water loss per day is approximately 1 1/2 liters higher than the drinking water intake figure would be.

"Human Designs" (11) by Beck (1971) indicates that between 2200 ml and 2800 ml are required for an average adult with an average 2500 ml daily fluid intake. This author, however, reverses the food and drink quantities shown above. Both of these references indicate that 1 cc of water is required per calorie of food intake.

Two articles relating to the fluid intake of children might be cited here. One, by Galagan, et al (12), used children from under one year of age to age ten and showed that total fluid intake per pound of body weights was highest among infants and decreased with age. The water intake listed average 0.40 ounces (12 ml) per day per pound of body weight. They also found that water intake increased directly with increases in temperature.

The second article by Bonham, et al (13) concerns six-year old children and lists 0.70 ounces (21 ml) per day per pound. This is total fluid and includes milk. If a child of this age weighed 50 lbs., he would drink about one liter per day.

The "Bioastronautics Data Book" (14) lists an average of 2400 ml total water intake but indicates the breakdown as 1,500 ml from drinking water, 600 ml from food and 30 ml from oxidation of food.

More recently, the Task Group on Reference Man (1974)(15) estimated the water-based fluid intake of an adult man to be 1650 ml/day, with corresponding values for an adult woman of 1200 ml/day and for a child of 950 ml/day.

Considering all the information we have available, two liters per day drinking water consumption for the average man should be a reasonable estimate. It is twice the amount listed by some authors and 30% higher than other authors list as an average figure and is therefore defensible as a reference standard.

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ARSENIC

The high toxicity of arsenic and its widespread occurrence in the environment necessitate the setting of a limit on the concentration of arsenic in drinking water.

The presence of arsenic in nature is due mainly to natural deposits of the metalloid and to its extensive use as a pesticidal agent. Arsenic concentrations in soils range from less than one part per million (mg/l) to several hundred mg/l in those areas where arsenical sprays have been used for years. Despite relatively high concentrations of arsenic in soils, plants rarely take up enough of the element to constitute a risk to human health (1, 2). Despite the diminishing use of arsenicals as pesticides, presently several arsenites are used as herbicides and some arsenates as insecticides. In 1964, farmers in the U.S. used a combined total of approximately 15 million pounds of arsenicals (3).

The chemical forms of arsenic consist of trivalent and pentavalent inorganic compounds and trivalent and pentavalent organic agents. It is not known which forms of arsenic occur in the drinking water. Although combinations of all forms are possible, it can be reasonably assumed that the pentavalent inorganic form is the most prevalent. Conditions that favor chemical and biological oxidation promote the shift to the pentavalent specie; and conversely, those that favor reduction will shift the equilibrium to the trivalent state.

The population is exposed to arsenic in a number of ways. Arsenic is still used, albeit infrequently, to treat leukemia, certain types of anemia, and certain skin diseases (4). In the diet, vegetables and grain contain an average of 0.44 ppm and meats an average of 0.5 ppm of arsenic (5). Organic arsenicals are deliberately introduced into the diet of poultry and pigs as growth stimulators and pesticides. The Food and Drug Administration has set tolerance limits for residues of arsenicals on fruits and vegetables (3.5 mg as As_2O_3 per kg) and in meat (0.5 to 2.0 mg as As per kg) (6). Shellfish are the dietary components that usually contain the highest concentrations of arsenic, up to 170 mg/kg (2, 7, 8).

For the entire U.S., the arsenic concentrations in air range from a trace to 0.75 ug/m^3 (9). Airborne arsenic is usually the result of operating cotton gins, manufacturing arsenicals, and burning coal.

Arsenic content of drinking water ranges from a trace in most U.S. supplies to approximately 0.1 mg/l (10). No adverse health effects have been reported from the ingestion of water containing 0.1 mg/l of arsenic.

The toxicity of arsenic is well known, and the ingestion of as little as 100 mg can result in severe poisoning. In general, inorganic arsenicals are more toxic to man and experimental animals than the organic analogs; and arsenic in the pentavalent state is less toxic than that in the trivalent form.

Inorganic arsenic is absorbed readily from the gastro-intestinal tract, the lungs, and to a lesser extent from the skin, and becomes distributed throughout the body tissues and fluids (4). Inorganic arsenicals appear to be slowly oxidized in vivo from the trivalent to the pentavalent state; however, there is no evidence that the reduction of pentavalent arsenic occurs within the body (5, 11-13). Inorganic arsenicals are potent inhibitors of the intracellular sulfhydryl (-SH) enzymes involved in cellular oxidations (14). Arsenic is excreted via urine, feces, sweat, and the epithelium of the skin (15-20). A single dose is usually excreted largely in the urine during the first 24 to 48 hours after administration; but elimination of the remainder of the dose continues for 7 to 10 days thereafter. During chronic exposure arsenic accumulates mainly in bone, muscle, and skin, and to a smaller degree in liver and kidneys. After cessation of continuous exposure, arsenic excretion may last up to 70 days (14).

A number of chronic oral toxicity studies with inorganic arsenite and arsenate (21-25) demonstrated the minimum-effect and no-effect levels in dogs, rats, and mice. Three generations of breeding mice were exposed to 5 ppm of arsenite in the diet with no observable effects on reproduction. At high doses (i.e., 200 mg/l or greater) arsenic is a physiological antagonist of thyroid hormones in the rat (26). Arsenic is also an antagonist of selenium and has been reported to counteract the toxicity of seleniferous foods when added to argicultural animals' feed water (27, 28). Rats fed shrimp meat containing a high concentration of arsenic retain very little of the element as compared to rats fed the same concentrations of either arsenic trioxide or calcium arsenate (29), suggesting that the arsenic in shellfish tissues may be less toxic to mammals than that ingested in other forms.

In man, subacute and chronic arsenic poisoning may be insidious and pernicious. In mild chronic poisoning, the only symptoms present are fatigue and loss of energy. The following symptoms may be observed in more severe intoxication: gastrointestinal catarrh, kidney degeneration, tendency to edema, polyneuritis, liver cirrhosis, bone marrow injury, and exfoliate dermatitis (30, 31). In 1962, thirty-two school-age children developed a dermatosis associated with cutaneous exposure to arsenic trioxide (32, 33). It has been claimed that individuals become tolerant to arsenic. However, this apparent effect is probably due to the ingestion of the relatively insoluble, coarse powder, since no true tolerance has ever been demonstrated (14).

Since the early nineteenth century, arsenic was believed to be a carcinogen; however, evidence from animal experiments and human experience has accumulated to strongly suggest that arsenicals do not produce cancer. One exception is a report from Taiwan showing a dose-response curve relating skin cancer incidence to the arsenic content of drinking water (44). Some reports incriminated arsenic as a carcinogen (34, 35), but it was later learned that agents other than the metalloid were responsible for such cancers (36). Sommers and McManus (37) reported several cases of cancer in individuals who had at some time in their lives been exposed to therapeutic doses of arsenic trioxide (usually in Fowler's Solution). Patients displayed characteristic arsenic keratosis, but there was no direct evidence that arsenic was the etiologic agent in the production of the carcinoma.

Properly controlled studies (38, 39) have demonstrated that industrial workers do not have an increased prevalence of cancer despite continued exposure to high concentrations of arsenic trioxide. In the study by Pinto and Bennett (39), the exposure was estimated by comparing the arsenic excreted in urine of control and exposed populations. In the experimental group, some workers who had been exposed to arsenic trioxide for up to 40 years, excreted 0.82 mg of arsenic per liter, or more than six times the concentration of the control population. In addition, attempts to demonstrate through animal studies that arsenic is tumorigenic have met with failure (23, 35, 40-42). The possible co-carcinogenic role of arsenic trioxide in the production of methylcholanthrene-induced skin tumors has been investigated and found to have no significant effect (43).

However, some recent evidence supports the view that arsenic is carcinogenic. Industrial workers in a plant manufacturing arsenic powder were exposed to arsenic dust and showed a higher incidence of skin and lung cancer than other occupational groups (44, 45, 46). Ulceration of the nasal septum appears to be a common finding among workers exposed to inorganic arsenic. The incidence of skin cancer has also been reported to be unusually high in areas of England where arsenic was present in drinking water at a level of 12 mg/l (47). More recently Lee and Fraumeni found that the mortality rate of white male smelter workers exposed to both arsenic trioxide and sulfur dioxide exceeded the expected mortality rate by a statistically significant margin and found that lung cancer deaths among these workers increased with increasing lengths of exposure to arsenic trioxide. They concluded that their findings were "consistent with the hypothesis that exposure to high levels of arsenic trioxide, perhaps in interaction with sulfur dioxide or unidentified chemicals in the work environment, is responsible for the three-fold excess of respiratory cancer deaths among smelter workers" (48).

Similarly, Ott, et al., found, in a study for the Dow Chemical Company, that exposed employees in a dry arsenical manufacturing plant experienced a three-fold increase in lung cancer over the rate for non-exposed employees (49).

Baetjer, et al., in a study for the Allied Chemical Company, found that 19 of the 27 deaths occuring in this population between 1960 and 1972 were due to cancer as compared to an expected number, based on figures adjusted for age, race, and sex, of 7.3 cancer-related deaths (50).

Additional medical problems relating to arsenic content of drinking water have been reported from several other countries. Several epidemiological studies in Taiwan (51-55) have reported the correlation between increased incidence of hyperkeratosis and skin cancer with the consumption of water with arsenic content higher than 0.3 mg/l. A similar problem has been reported in Argentina (56-58). Dermatological manifestations of arsenicism were noted in children of Antofagasto, Chile, who used a water supply with 0.8 mg/l. A new water supply was provided, and preliminary data show that arsenic levels of hair have decreased, and further study will be made of the health of persons born since the change in supply (59). Arsenicism affecting two members of a family where the arsenic content of the family's well varied between 0.5 and 2.75 mg/l over a period of several months, was reported in Nevada (60). A study in California found that a greater proportion of the population had elevated concentrations of arsenic in the hair when the drinking water had more than 0.12 mg/l than when it was below this concentration, but illness was not noted (61). In none of the cited incidents of apparent correlation of arsenic in drinking water with increased incidence of hyperkeratosis and skin cancer has there been any confirmed evidence that arsenic was the etiologic agent in the production of carcinomas.

Arsenic is a geochemical pollutant, and when it occurs in an area it can be expected to be in the air, food, and water, but in other cases it is due to industrial pollution. In some epidemiological studies it is difficult to determine which exposure is the greater problem. A recent study (62) of metallic air pollutants showed that arsenic levels of hair were related to exposure from this source, but other exposures were not quantitated. The Taiwan studies were able to compare quite similar populations that differed only in the water intake. Deep wells contained arsenic, but persons using shallow wells were not exposed.

The change in water supply in Chile provided a unique experience to demonstrate the effect of arsenic in drinking water in spite of other arsenic exposures.

It is estimated that the total intake of arsenic from food is an average of 900 µg/day (5). At a concentration of 0.05 mg per liter and an average intake of 2 liters of water per day, the intake from water would not exceed 100 µg per day, or approximately 10 percent of the total ingested arsenic.

In light of our present knowledge concerning the potential health hazard from the ingestion of arsenic, the concentration of arsenic in the drinking water shall not exceed 0.05 mg/l.

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BARIUM

Barium is recognized as a general muscle stimulant, including especially the heart muscle (1). The fatal dose for man is considered to be from 0.8-0.9 g as the chloride (550-600 mg Ba). Most fatalities have occurred from mistaken use of barium salts incorporated in rat poison. Barium is capable of causing nerve block (2) and in small or moderate doses produced transient increase in blood pressure by vasoconstriction (3). Aspirated barium sulfate has been reported to result in granuloma of the lung (4) and other sites in man (5). Thus, evidence exists for high acute toxicity of ingested soluble barium salts, and for chronic irreversible changes in tissues resulting from the actual depostion of insoluble forms of barium in sufficient amounts at a localized site. On the other hand, the recent literature reports no accumulation of barium in bone, muscle, or kidney from experimentally administered barium salts in animals (6). Most of the administered dose appeared in the liver with far lesser amounts in the lungs and spleen. This substantiates the prior finding of no measurable amounts of barium in bones or soft tissues of man (7). Later, more accurate analysis of human bone (British) showed 7 ug Ba/g ashed sample (8), but no increase in bone barium occurred from birth to death. Small amounts of barium have been shown to go to the skeleton of animals when tracer amounts of barium-140 were used (9), but no determinations of barium have been made in animals to which barium had been repeatedly administered for long periods.

No study appears to have been made of the amounts of barium that may be tolerated in drinking water or of effects from prolonged feeding of barium salts from which an acceptable water **guideline** may be set. A rational basis for a water guideline may be derived from the threshold limit of 0.5 mg Ba/m³ air set by the American Conference of Governmental Industrial Hygienists (10) by procedures that have been discussed (11). By assuming that 75% of the barium inhaled is absorbed into the blood stream and that 90% is a reasonable factor for absorption via the gastrointestinal tract, a value of 2 mg/l can be derived as an approximate limiting concentration for a healthy adult population. The introduction of a safety factor to account for heterogeneous populations results in the derivation of 1 mg/l as a limit that should constitute a "no effect" level in water. Because of the seriousness of the toxic effects of barium on the heart, blood vessels, and nerves, drinking water shall not contain barium in a concentration exceeding 1 mg/l.

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CADMIUM

As far as is known, cadmium is biologically a nonessential, nonbeneficial element of high toxic potential. **Evidence for the serious** toxic potential of cadmium is provided by: (a) poisoning from cadmium-contaminated food (1) and beverages (2); (b) epidemiologic evidence that cadmium may be associated with renal arterial hypertension under certain conditions (3); (c) epidemiologic association of cadmium with "Itai-itai" disease in Japan (4); and (d) long-term oral toxicity studies in animals.

The possibility of cadmium being a water contaminant has been reported in 1954 (5); seepage of cadmium into ground water from electroplating plants has resulted in cadmium concentrations ranging from 0.01 to 3.2 mg/l. Other sources of cadmium contamination in water arise from zinc-galvanized iron in which cadmium is a contaminant. The average concentration of cadmium in drinking water from community supplies is 1.3 ug per liter in the United States. Slight amounts are common, with 63 percent of samples taken at household taps showing 1 ug per liter or more. Only 0.3 percent of tap samples would be expected to exceed the limits of 10 ug per liter (6).

Several instances have been reported of poisoning from eating substances contaminated with cadmium. A group of school children were made ill by eating popsicles containing 13 to 15 mg/l cadmium (1).

This is commonly considered the emetic threshold concentration for cadmium. It has been stated (7) that the concentration and not the absolute amount determines the acute cadmium toxicity; equivalent concentrations of cadmium in water are likewise considered more toxic than equivalent concentrations in food probably because of the antagonistic effect of components in the food.

Chronic oral toxicity studies in rats, in which cadmium chloride was added to various diets at levels of 15, 45, 75, and 135 ppm cadmium, showed marked anemia, retarded growth, and in many instances death at the 135 ppm level. At lower cadmium levels, anemia developed later; only one cadmium-fed animal had marked anemia at the 15 ppm level. Bleaching of the incisor teeth occurred in rats at all levels, except in some animals at 15 ppm. A low protein diet increased cadmium toxicity. A maximal "no effect" level was thus not established in the above studies (8). A dietary relation to cadmium toxicity has been reported by others (9).

Fifty mg/l of cadmium administered as cadmium chloride in food and drinking water to rats resulted in a reduction of blood hemoglobin and lessened dental pigmentation. Cadmium did not decrease experimental caries (10).

In a study specifically designed to determine the effects of drinking water contaminated with cadmium, five groups of rats were exposed to drinking water containing levels from 0.1 to 10 mg/l. Although no effects of cadmium toxicity were noted, the content of cadmium in the kidney and liver increased in direct proportion to the dose at all levels including 0.1 mg/l. At the end of one year, tissue concentrations approximately doubled those at six months. Toxic effects were evident in a three-month study at 50 mg/l (11). Later work has confirmed the virtual absence of turnover of absorbed cadmium (12). More recently, the accumulation of cadmium in renal and hepatic tissue with age has been documented in man (13).

Recent epidemiological evidence strongly suggests that cadmium ingestion is associated with a disease syndrome referred to as "Itai-itai" in Japan (4). The disease syndrome is characterized by decalcification of bones, proteinuria, glycosuria and increased serum alkaline phosphatase, and other more subjective symptoms. Similar clinical manifestations have been noted in cadmium workers (14). Yamagatta and Shigematsu (15) have estimated the current daily intake of cadmium in an endemic "Itai-itai" area as 600 µg. The authors from a geological and topographical survey as well as knowledge of local customs, concluded that the daily cadmium intake in the endemic area was probably higher in the past. They concluded that 600 ug per day would not cause "Itai-itai" disease. The average ingestion of cadmium is 59 µg/day in non-polluted areas of Japan.

The association of cardiovascular disease, particularly hypertension, with ingestion of cadmium remains unsettled. Conflicting evidence has been found both in man (3, 16) and in animals (17, 18). It is notable that hypertension has not been associated with 'Itai-itai" disease (19).

The main sources of cadmium exposure in the United States to the general population appear to be the diet and cigarette smoking. R. E. Duggan and P. E. Corneliussen (20) of the FDA in a market basket survey of five geographic regions in the U. S. found the "daily intake" of cadmium to be 50 µg in 1969 and 30 µg in 1970. Each market basket represented a 2-week diet constructed for a 16-19 year-old male. Murthy and associates found the cadmium intake of children to be 92 µg per day from a study of institutional diets (21). Other estimates are also generally higher than FDA's -- ranging from 67 to to 200 µg/day. A review of these data suggest 75 µg as a reasonable estimate of average daily dietary intake (22, 23, 24, 25).

Cigarette smoking has also been shown to be important. Twenty cigarettes per day will probably cause the inhalation of 2-4 µg of cadmium (26). However, the absorption rate associated with cigarette smoke inhalation is much larger than that associated with food ingestion. Lewis (27) has shown in autopsy studies that men who smoke one or more packages of cigarettes per day have a mean cadmium concentration in the renal cortex (wet weight) double the level in a control group of non-smokers. Hammer (24) in similar studies also found renal wet weight concentrations for those smoking 1 1/2 or more packages of cigaretts per day to be more than twice as high as for non-smokers. In terms of effective body burden, then, cigarette smoking may double the level derived from food intake alone.

Exactly what exposure to cadmium will cause proteinuria, the earliest manifestation of chronic cadmium poisoning, is unknown. From animal experiments and very limited human observation in cases of industrial exposure, it is believed that a cadmium level of 200 ppm wet weight in the renal cortex will be associated with proteinuria. (However, it should be noted that in one case a level of 446 ppm was found by Axelsson and Piscator without proteinuria) (29). It has been estimated that with 5% gastrointestinal absorption, rapid excretion of 10% of the absorbed dose, and 0.05% daily excretion of the total body burden, it would take 50 years with a daily ingestion of 352 µg of Cd to attain the critical level of 200 ppm wet weight in the renal cortex. The percentage absorption in man is unknown. If the gastrointestinal absorption of cadmium in man really is about 3%, it would probably take about 500-600 µg ingested per day to cause proteinuria.

Concentration of cadmium shall be limited to 0.010 mg/l in drinking water. At this level it would contribute 20 µg per day to the diet of a person ingesting 2 liters of water per day. Added to an assumed diet of 75 µg/day, this would provide about a four-fold safety factor. This does not, however, take cigarette smoking into account.

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CHROMIUM

Chromium, particularly in the hexavalent state, is toxic to man, produces lung tumors when inhaled, and readily induces skin sensitizations. Chromium occurs in some foods, in air including cigarette smoke, and in some water supplies (see Table I). It is usually in an oxidized state in chlorinated or aerated waters, but measurements for total chromium are easily made by atomic absorption, so the somewhat conversative total value is used for this guideline.

TABLE I

U.S. urban air concentrations range, 1965 (1)	0-0.028 µg/m^3
Chromium content in cigarette tobacco (2)	1.4 µg/cigarette
Chromium in foods cooked in stainless-steel ware (3)	0-0.35 mg/100 g
Chromium concentration range in water supplies 1969 (4)	0-0.08 mg/l

Comparatively little data are available on the incidence and frequency of distribution of chromium in foods. Although most information has limited applicability, one study (5) determined the occurrence of chromium and other elements in institutional diets. In that investigation, the concentrations of chromium in foods ranged from 0.175 to 0.470 mg/kg.

Chromium has not been proved to be an essential or a beneficial element in the body. However, some studies suggest that chromium may indeed by essential in minute quantities (5,6,7). At present, the levels of chromium that can be tolerated by man for a lifetime without adverse effects on health are still undetermined. A family of four individuals is known to have drunk water for periods of 3 years at a level as high as 0.45 milligrams chromium per liter without known effects on their health, as determined by a single medical examination (8).

A study by MacKenzie et al (8) was designed to determine the toxicity to rats of chromate (Cr^{+6}) and chromic (Cr^{+3}) ion at various levels in the drinking water. This study showed no evidence of toxic responses after one year at levels from 0.45 to 25 mg/l by the tests employed, viz., body weight, food consumption, blood changes, and mortality. Significant accumulation of chromium in the tissues occurred abruptly at concentrations above 5 mg/l; however, no study has been made of the effects of chromium on a cancer-susceptible strain of animal. Recent studies demonstrated that 0.1 mg of potassium dichromate per kg enhances the secretory and motor activity of the intestines of the dog (10).

From these and other studies of toxicity (11-15), it would appear that a concentration of 0.05 mg/l of chromium incorporates a reasonable factor of safety to avoid any hazard to human health.

In addition, the possibility of dermal effects from bathing in water containing 0.05 mg/l would likewise appear remote, although chromium is recognized as a potent sensitizer of the skin (3). Therefore, drinking water shall not contain more than 0.05 mg/l of chromium.

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CYANIDE

Cyanide in reasonable doses (10 mg or less) is readily converted to thiocyanate in the human body and is thus much less toxic for man than fish. Usually, lethal toxic effects occur only when the detoxifying mechanism is overwhelmed. The oral toxicity of cyanide for man is shown in the following table.

Oral Toxicity of Cyanide for Man

Dosage	Response	Literature Citations
2.9-4.7 mg/l	Noninjurious	(1)
10 mg, single dose	Noninjurious	(2)
19 mg/l in water	Calculated from threshold limited for air to be safe	(3)
50-60 mg, single dose	Fatal	(4)

Proper chlorination to a free chlorine residual under neutral or alkaline conditions will reduce cyanide to very low levels. The acute oral toxicity of cyanogen chloride, the chlorination product of hydrogen cyanide, is approximately one-twentieth that of hydrogen cyanide (5). It should be noted that at a pH of 8.5 cyanide is readily converted to cyanate which is much less toxic.

Because of the above considerations, and because cyanide occurs, however rarely, in drinking water primarily as the result of spills or other accidents, there appears to be no justification for establishing a maximum contaminant level for cyanide.

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FLUORIDE

The Food and Nutrition Board of the National Research Council has stated that fluoride is a normal constituent of all diets and is an essential nutrient (1). In addition, fluoride in drinking water will prevent dental caries. When the concentration is optimum, no ill effects will result, and the caries rate will be 60-65 percent below the rates in communities with little or no fluoride (2,3).

Excessive fluoride in drinking water supplies produces objectionable dental fluorosis which increases with increasing fluoride concentration above the recommended upper control limits. In the United States, this is the only harmful effect observed to result from fluoride found in drinking water (4,5,6,7,8,9,10,11). Other expected effects from excessively high intake levels are: (a) bone changes when water containing 8-20 mg fluoride per liter (8-20 mg/l) is consumed over a long period of time (7); (b) crippling fluorosis when 20 or more mg of fluoride from all sources is consumed per day for 20 or more years (12); (c) death when 2,250-4,500 mg of fluoride (5,000-10,000 mg sodium fluoride) is consumed in a single dose (7).

The optimum fluoride level (see Table 1) for a given community depends on climatic conditions because the amount of water (and consequently the amount of fluoride) ingested by children is primarily influenced by air temperature. This relationship was first studied and reported by Galagan and Associates in the 1950's (13,14,15,16), but has been further investigated and supported by Richards, et al (17) in 1967. The control limits for fluoride supplementation, as shown in Table 1, are simply the optimum concentrations for a given temperature zone, as determined by the Public Health Service, DHEW, from the data cited, plus or minus 0.1 mg/liter.

Many communities with water supplies containing less fluoride than the concentration shown as the lower limit for the appropriate air temperature range have provided fluoride supplementation (18, 19, 20, 21). Other communities with excessively high natural fluoride levels have effectively reduced fluorosis by partial defluoridation and by change to a water source with more acceptable fluoride concentration (22, 23, 24).

Richards, et al (17) reported the degree of fluorosis among children where the community water supply fluoride content was somewhat above the optimum value. From such evidence, it is apparent that an approval limit (see Table 1) slightly higher than the optimum range can be tolerated without any mottling of teeth, so where fluorides are native to the water supply, this concentration is acceptable. Higher levels should be reduced by treatment or blending with other sources lower in fluoride content. In such a case, the optimum value should be sought and maintained.

Table 1

Annual Average of Maximum Daily Air Temperatures F	Recommended Control Limits Fluoride Concentrations in mg/l Lower	Optimum	Upper	Approval Limit mg/l
50.0 - 53.7	1.1	1.2	1.3	2.4
53.8 - 58.3	1.0	1.1	1.2	2.2
58.4 - 63.8	0.9	1.0	1.1	2.0
63.9 - 70.6	0.8	0.9	1.0	1.8
70.7 - 79.2	0.7	0.8	0.9	1.6
79.3 - 90.5	0.6	0.7	0.8	1.4

It should be noted that, when supplemental fluoridation is practiced, it is particularly advantageous to maintain a fluoride concentration at or near the optimum. The reduction in dental caries experienced at optimal fluoride concentrations will be diminished by as much as 50% when the fluoride concentration is 0.2 mg/l below the optimum. (25,26)

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LEAD

Lead is well known for its toxicity in both acute and chronic exposures. Kehoe (1) has pointed out that in technologically developed countries, the widespread use of lead multiplies the risk of exposure of the population to excessive lead levels. For this reason, the necessity of constant surveillance of the lead exposure of the general population via food, air, and water is imperative.

The clinical picture of lead intoxication has been well documented (2). Unfortunately, the general picture of the symptoms is not unique (i.e., gastrointestinal disturbances, loss of appetite, fatigue, anemia, motor nerve paralysis, and encephalopathy) to lead intoxication and often this has resulted in misdiagnosis (3, 4). Several laboratory tests that are sensitive to increased lead blood levels have been developed for diagnostic purposes, but their relationship to the effects of lead intoxication are incompletely understood. The most sensitive of these is the inhibition of red cell-aminolevulinic acid dehydrase (ALAD) which correlates well with blood lead levels from 5-95 μg/100 g blood (5, 6). Because this is not the rate-limiting step in porphyrin biosynthesis, accumulation of aminolevulinic acid (ALA) does not occur until high blood lead levels are reached. Other such tests, which correlate with blood lead to a lesser degree and at higher levels, are the measurement of urinary coproporphyrins, the number of coarsely stippled red blood cells and the basophilic quotient (6). These changes, in themselves, have little known significance in terms of the danger to the health of the normal individual, for although red cell life-time can be shown to decrease (7), high lead concentrations are required for the development of the anemia typical of lead intoxication (8). Urinary ALA, however, has been shown to be closely related to elevated lead levels in soft tissues (9, 10) and is considered to be indicative of a probable health risk (11).

Young children present a special case in lead intoxication, both in terms of the tolerated intake and the severity of the symptoms (8). Lead encephalopathy is most common in children up to three years of age (12). The most prevalent source of lead in these cases of childhood poisoning has been lead-containing paint still found in many older homes (1, 12). Prognosis of children with lead encephalopathy is poor, with or without treatment. Up to 94% of the survivors have been found to have psychological abnormalities (13). It is still unknown whether smaller intakes of lead without encephalopathy or subclinical lead poisoning causes mental retardation or psychological abnormalities. Several studies in man and animals suggest this, (14, 15, 16, 17), but a well-controlled prospective study in man has yet to be done. ALAD in baby rats' brains is suppressed by excess lead (18); however, the significance of this finding to humans is unknown. Some groups of individuals who experienced lead intoxication at an early age and survived have demonstrated a high incidence of chronic nephritis in later life (19). Recent work has demonstrated a high incidence of aminoaciduria and other biochemical changes of kidney disease in children in Boston with excessive lead exposure (17). A recent study found anemia in children with blood levels from 37-60 μg/100 ml to be common (20). There is evidence that lead in high doses in animals affects the immunological system (21, 22, 23, 24); this, however, has not yet been demonstrated in man.

The average daily intake of lead via the diet was 0.3 mg in 1940 (25) and rarely exceeded 0.6 mg. Data obtained subsequent to 1940 indicate that the intake of lead appears to have decreased slightly since that time (1, 26). Inhaled lead contributes about 40% to total body burden of lead (1, 27) in the average population. Cigarette smoking in some studies in the past has also been associated with slightly elevated blood lead levels (3).

Accumulation of lead with age in non-occupationally exposed individuals has been demonstrated (26, 28, 29). The bulk of this lead distributes to bone, while soft tisssue levels vary only slightly from normal even with high body burdens (30). Blood levels vary only slightly from normal even with high body burdens (30). Blood levels of lead in persons without unusual exposure to lead range up to 40 μg/100 g and average about 26 μg/100 g (1). The U.S. Public Health Service (31) considers

40 µg/100 g lead or over in whole blood in older children and adults on two separate occasions as evidence suggestive of undue absorption, either past or present. Levels of 50-79 µg/100 g require immediate evaluation as a potential poisoning case. Eighty µg/100 g or greater is considered to be unequivocal lead poisoning. The 40 µg/100 g lead level in blood probably has a biological effect as the National Academy of Science Lead Panel (11) concluded:

> "...the exponential increase in ALA excretion associated with blood lead content above approximately 40 µg/100 g of blood signifies inhibition of ALAD that is significant physiologically in vivo."

In addition animal experiments show beginning renal injury at about the same exposure level causing urinary ALA increase (32).

Blood lead is increased in urban vs. surburban (28,33,34), near to vs. distant from large motorways (35,36) and in occupational exposure to areas of high traffic density (37,38,39). Lead in soil has epidemiologically been implicated in increased blood lead in children (40).

The World Health Organization Committee (41), assuming 10% of lead from food and water is absorbed, established in adults a "Provisional tolerable weekly intake" of 3 mg of lead per person (the maximum lead exposure the average person can tolerate without increased body burden). (Kehoe considers 600 µg per day the limit).

Assuming 10% absorption from the gastrointestinal tract, approximately 40 ug of lead per day would be absorbed, by the WHO standard. With the average diet containing 100-300 ug lead per day, and the average urban air containing 1 to 3 ug/m^3 of air, the average urban man would absorb 16 to 48 ug of lead per day. (The contribution from 1 ug/m^3 lead in air at 20 m respiratory volume with 30% absorption is 6 ug). Just from food and air alone, some urban dwellers would have excessive exposure by the WHO standard. Urban children are further exposed by dust with levels of over 1000 ug/g (40, 42, 43) and because airborne lead particles vary in density inversely from the distance from the ground (44, 45). Rural children have significantly less exposure than do urban children to these sources. Additionally, children have increased risk, because they have food and air intakes proportionally greater than their size and they might absorb a larger percentage from their gut, possibly 50% of ingested lead (46). Lead might also have a greater effect on their developing neurological, hematological, and immunological systems (18, 20-24, 47, 48). Likewise, fetuses of mothers unduly exposed may be at risk (49, 51), and McIntire concluded that there is a definite fetal risk maximal in the first trimester from intrauterine exposure to increased lead in maternal blood (52).

The lead concentrations in finished water ranged from 0 to 0.64 mg/liter in the Community Water Supply Study conducted in 1969 (53). Of the 969 water supplies surveyed, 1.4% exceeded 0.05 mg/liter of lead in drinking water. Five of the water supplies in this sample had sufficient lead to equal or exceed the estimated maximum safe level of lead intake (600 µg/day) without considering the additional contribution to the total intake by other routes of exposure. Under certain conditions, (acidic soft water, in particular) water can possess sufficient plumbosolvency to result in appreciable concentrations of lead in water standing in lead pipes overnight (54).

As a result of the narrow range between the lead exposure of the average American in everyday life and exposure which is considered excessive (especially in children) it is imperative that lead in water be maintained within rather strict limits. Since a survey (55) of lead in surface water of the U.S.A. and Puerto Rico found only 3 of 726 surface waters to exceed 0.05 mg/l; the standard of 0.05 mg/l should be obtainable. For a child one to three years old drinking one liter of water a day (probably the most a child would drink), the contribution would be 0.05 mg/l x 1.0 liter equals 0.050 mg. The diet is estimated by scaling down the average adult's diet to be 150-200 µg (56). Assuming the fraction of lead absorbed is the same for lead in food and water, water would contribute 25 to 33% of the lead normally ingested. For an adult drinking 2 liters per day, the contribution would be 0.1 mg/0.3 mg, or 33% of food. At lower concentrations, for example, 0.015 mg/l, the average concentration in drinking water, the contribution of water in an adult or child would be less than 10% of that of food.

It should be reemphasized that the major risk of lead in water is to small children (50). The potentially significant sources of lead exposure to children which have been documented include paint, dust (40, 42, 43), canned milk (58, 59), tooth paste (60, 61), toys, newsprint ink (62, 63), and air. Although paint is most strongly implicated epidemiologically, there is growing evidence that others, such as dust, are important (40). There is a serious problem with excess lead in children; it is well documented. It can lead to lead poisoning. Lead poisoning does cause death and morbidity in children. A survey of 21 screening programs (64) testing 344,657 children between 1969 and 1971 found 26.1% or over 80,000 children with blood leads of over 40 μg/l (which is considered evidence of excessive exposure.) Several recent studies suggest that the frequency of intellectual and psychological impairment is increased among children overexposed to lead who were not thought to have had overt clinical lead poisoning (14, 15, 16, 17). With the widespread prevalence of undue exposure to lead in children, its serious potential sequelae, and studies suggesting increased lead absorption in children (chronic brain or kidney damage, as well as acute brain damage); it would seem wise at this time to continue to limit the lead in water to as low a level as practicable. Data from the Community Water Supply Study and other sources indicate that a lead concentration of 0.05 mg/l or less can be attained in most drinking water supplies. Experience indicates that less than four percent of the water samples analyzed exceed the 0.05 mg/l limit and the large majority of these are due to stability (corrosion) problems not due to naturally occurring lead content in the raw waters.

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MERCURY

Environmental exposure of the population to mercury and its compounds poses an unwarranted threat to man's health. Since conditions indicate an increasing possibility that mercurials may be present in drinking water, there is a need for a guideline that will protect the health of the water consumer.

Mercury is distributed throughout the environment. And as a result of industrial and agricultural applications, large increases in concentrations above natural levels in water, soils, and air may occur in localized areas around chlor-alkali manufacturing plants and industrial processes involving the use of mercurial catalysts, and from the use of slimicides primarily in the paper-pulp industry and mercurial seed treatment.

Mercury is used in the metallic form, as inorganic mercurous (monovalent) and mercuric (divalent) salts, and in combination with organic molecules (viz. alkyl, alkoxyalkyl, and aryl).

The presence of mercury in fresh and sea water was demonstrated more than 50 years ago (1-4). In early studies in Germany, Stock (5,6) found mercury in tap water, springs, rain water, and beer. In all water, the concentration of mercury was consistently less than one µg/l; however, beer occasionally contained up to 15 µg/l. A recent survey (7) demonstrated that most U.S. streams and rivers contain 0.1 µg of dissolved mercury or less per liter.

Presently the concentration of mercury in air is ill-defined for lack of analytical data. In one study (8) the concentration of mercury contained in particulates in the atmosphere of 2 U.S. cities was measured and ranged from 0.03 to 0.21 $\mu g/m^3$. One review (9) cited values up to 41 $\mu g/m^3$ of particulate mercury in one U.S. metropolitan area.

Outside of occupational exposure, food, particularly fish, is the greatest contributor to body burden of mercury. In 1967 a limited study of mercury residues in foods was conducted, involving 6 classes of foods. The results indicated levels of mercury in the order of 2 to 50 µg/kg. The Atomic Energy Commission sampled various foods for mercury in its tri-city study and reported levels between 10 and 70 µg of mercury per kg of meats, fruits, and vegetables. In 1970, it was discovered that several types of fresh and salt water fish contained mercury (mostly in the alkyl form) in excess of the FDA guideline of 0.5 ppm (500 µg/kg). Mercury in bottom sediments had been converted by micro-organisms to the alkyl form, entered the food chains, and had accumulated in the higher members of the chains. Game birds were also discovered to have high levels of mercury in their tissues, persumably from the ingestion of mercury-treated seeds or of smaller animals that had ingested such seeds. The Food and Drug Administration has established a guideline of 0.5 ppm for the maximum allowable concentration of mercury in fish for human consumption; but for all other foodstuffs, no tolerances have been established.

Mercury poisoning may be acute or chronic. Generally mercurous salts are less soluble than mercuric salts and are consequently less toxic acutely. Acute intoxication is usually the result of suicidal or accidental exposure. For man the fatal oral dose of mercuric salts ranges from 20 mg to 3 g. The acute syndrome consists of an initial phase referable to local effects (viz. pharyngitis, gastroenteritis, vomiting, and bloody diarrhea) followed later by symptoms of systemic poisoning (viz. anuria with uremia, stomatitis, ulcerative-hemorrhagic colitis, nephritis, hepatitis, and circulatory collapse) (10).

Acute intoxication from the inhalation of mercury vapor or dusts leads to the typical symptoms of mercury poisoning coincident with lesions of the mucous membranes of the respiratory tract which may ultimately develop into bronchitis and bronchopneumonia. Inhalation of mercury in concentrations of 1,200 to 8,500 $\mu g/m^3$ results in acute intoxication (10). In severe cases, signs of delayed neurotoxic effects, such as muscular tremors and psychic disturbances, are observed. The Threshold Limit Value for all forms of mercury except alkyl is 0.05 mg/m^3 in the U.S. (11).

Chronic mercury poisoning results from exposure to small amounts of mercury over extended periods of time. Chronic poisoning from inorganic mercurials has been most often associated with industrial exposure, whereas that from the organic derivatives has been the result of accidents or environmental contamination.

Workers continually exposed to inorganic mercury are particularly susceptible to chronic mercurialism. Usually the absorption of a single large dose by such individuals is sufficient to precipitate the chronic disease that is characterized mainly by central nervous system toxicity (10, 12, 13). Initially, non-specific effects, such as headaches, giddiness, and reduction in the power of perception, are observed. Fine tremors gradually develop primarily in the hands and are intensified when a particular movement is begun. In prolonged and severe intoxication, fine tremor is interspersed with coarse, almost choreatic, movements. Excessive salivation, often accompanied by a metallic taste and stomatitis, is common. As the illness progresses, nervous restlessness (erethismus mercurialis) appears and is characterized by psychic and emotional distress and in some cases hysteria. Although the kidney is less frequently affected in this type of poisoning, chronic nephrosis is occasionally observed.

Several of the compounds used in agriculture and industry (such as alkoxyalkyls and aryls) can be grouped, on the basis of their effects on man, with inorganic mercury to which the former compounds are usually metabolized.

Alkyl compounds are the derivatives of mercury most toxic to man, producing illness from the ingestion of only a few milligrams (21, 24). Chronic alkyl mercury poisoning, also known as Minamata Disease, is an insidious form of mercurialism whose onset may appear after only a few weeks of exposure or may not appear until after a few years of exposure. Poisoning by those agents is characterized mainly by major neurological symptoms and leads to permanent damage or death. The clinical features in children and adults include numbness and tingling of the extremities, incoordination, loss of vision and hearing, and intellectual deterioration. Autopsy of the clinical cases reveals severe brain damage throughout the cortex and cerebellum. There is evidence to suggest that compensatory mechanisms of the nervous system can delay recognition of the disease even when partial brain damage exists.

Several episodes of alkyl mercury poisoning have been recorded. As early as 1865, two chemists became ill and died as a result of inhaling vapors of ethyl mercury (14). One of the largest outbreaks occurred in a village near Minamata Bay, Japan, from 1953 through 1960. At least 121 children and adults were affected (of whom 46 died) by eating fish containing high concentrations of methyl mercury (15). Of the population affected, 23 infants and children developed a cerebral palsy-like disease which was referred to as Congenital (or Fetal) Minamata Disease. Similarly, in 1964 and 1965, the disease was reported among 47 persons, 6 of whom died, in Niigata, Japan. Hunter *et al* (16) reported 4 cases of industrial intoxication from handling of these agents. In Guatemala, Iraq, Pakistan, and the United States, the human consumption of grain treated with alkyl mercurials for seed purposes has led to the poisoning of more than 450 persons, some of whom died (17-20).

The congenital (fetal) disease observed in Minamata and Niigata emphasize the devastating and insidious nature of these agents. Of particular significance are the facts that (1) the affected children had not eaten contaminated fish and shellfish, and (2) the mothers apparently were not affected although they had consumed some contaminated food. Exposure of the fetus to mercury via the placenta and/or the mother's milk is believed to be the etiologic basis for this disease, thus indicating the greater susceptibility of infants to alkyl mercury.

Absorption is a factor important in determining the toxicity of alkyl mercurials. Berglund and Berlin (21) estimated that methyl mercury is absorbed at more than a 90% rate via gastro-intestinal tract as compared with 2% mercuric ion (22). In addition, methyl mercury crosses the placenta into the fetus and achieves a 30% higher concentration in fetal erythrocytes than in maternal red blood cells (23). However, the fetal plasma concentration of mercury is lower than that of the mother. The rate of uptake of methyl mercury into the fetal brain is as yet unknown. Alkyl mercury can cross the blood-brain barrier more easily than other mercurials, so that brain levels of mercury are much higher after a dose of alkyl mercury than after a corresponding dose of any other mercurial.

Excretion is of equal importance in determining the health hazard. Unlike inorganic mercury, alkyl mercury is excreted mainly in the feces. After exposure to methyl mercury, approximately 4% of the dose is excreted within the first few days, and about 1% per day thereafter (24). The biological half-life of methyl mercury in man is approximately 70 days.

Safe levels of ingested mercury can be estimated from data presented in "Methyl Mercury in Fish" (24). From epidemiological evidence, the lowest whole-blood concentration of methyl mercury associated with toxic symptoms is 0.2 µg/g. This blood concentration can be compared to 60 µg Hg/g hair. These values, in turn, correspond to prolonged, continuous exposure at approximately 0.3 mg Hg/70 kg/day. By using a safety factor of 10, the maximum dietary intake should be 0.03 mg Hg/person/day (30 µg/70 kg/day). Although the safety factor is computed for adults, limiting ingestion by children to 30 ug Hg/day is believed to afford some, albeit smaller, degree of safety. If exposure to mercury were from fish alone, the limit would allow for a maximum daily consumption of 60 grams (420 g/week) of fish containing 0.5 mg Hg/kg. In a given situation, if the total daily intake from all sources, air, water, and food, is approaching 30 ug/person/day, the concentration of mercury and/or the consumption of certain foods will have to be reduced if a safety factor of 10 is to be maintained. Fortunately, since only a small fraction of the mercury in drinking water is in the alkyl form, the risk to health from waterborne mercury is not nearly so great as is the risk from mercury in fish. Also fortunately, mercury in drinking water seldom exceeds 0.002 mg/l. Drinking water containing mercury at the approval limit of 0.002 mg/l will contribute a total of 4 ug Hg to the daily intake, and will contribute less than 4 ng methyl mercury to the total intake. (Assuming that less than 0.1% of the mercury in water is in the methyl mercury form.) Since the Regulations approval limit is seldom exceeded in drinking water, the margin of safety gained from the restricted intake of mercury in drinking water can be applied to the total intake with minimal economic impact.

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NITRATE

Serious and occasionally fatal poisonings in infants have occurred following ingestion of well waters shown to contain nitrate (NO_3) at concentrations greater than 10 mg/l nitrate nitrogen. This has occurred with sufficient frequency and widespread geographic distribution to compel recognition of the hazard by assigning a limit to the concentration of nitrate in drinking water at 10 mg/l as nitrogen. This is about 45 mg/l of the nitrate ion.

Nitrate in drinking water was first associated in 1945 with a temporary blood disorder in infants called methemoglobinemia (1). Since then, approximately 2000 cases of this disease have been reported from North America and Europe, and about 7 to 8 percent of the infants died (2, 3, 4). Evidence in support of the limit for nitrate is given in detail by Walton (2) in a survey of the reported cases of nitrate poisoning of infants before 1951. The survey shows that no cases of poisoning were reported when the water contained less than 10 mg/l nitrate nitrogen. More recent surveys (3, 4) involving 467 and 249 cases tend to confirm these findings. Frequently, however, water was sampled and analyzed retrospectively and therefore the concentration of nitrate which caused illness was not really known. Many infants have drunk water when the nitrate nitrogen was greater than 10 mg/l without developing the disease. Many public water supplies in the United States have levels of nitrate that routinely exceed the standard, but only one case associated with a public water supply has been reported (5).

A basic knowledge of the development of the disease is essential to understanding the rationale behind protective measures. The development of methemoglobinemia, largely confined to infants less than three months old, is dependent upon the bacterial conversion of the relatively innocuous nitrate ion to nitrite. Nitrite then converts hemoglobin, the blood pigment that carries oxygen from the lungs to the tissues, to methemoglobin. Because the altered pigment can no longer transport oxygen, the physiologic effect of methemoglobinemia is that of oxygen deprivation, or suffocation.

The ingestion of nitrite directly would have a more immediate and direct effect on the infant because the bacterial conversion step in the stomach would be eliminated. Fortunately, nitrite rarely occurs in water in significant amounts, but waters with nitrite nitrogen concentrations over 1 mg/l should not be used for infant feeding. Waters with a significant nitrite concentration would usually be heavily polluted and would be unsatisfactory on a bacteriological basis as well.

There are several physiological and biochemical features of early infancy that explain the susceptibility of the infant less then three months of age to this disorder. First, the infant's total fluid intake per body weight is approximately three times that of an adult (6). In addition, the infant's incompletely developed capability to secrete gastric acid allows the gastric pH to become high enough (pH of 5-7) to permit nitrate-reducing bacteria to reside high in the gastrointestinal tract. In this location, the bacteria are able to reduce the nitrate before it is absorbed into the circulation (7). To further predispose the infant, the predominant form of hemoglobin at birth, hemoglobin F (fetal hemoglobin), is more susceptible to methemoglobin formation than the adult form of hemoglobin (hemoglobin A) (8). Finally, there is decreased activity in the enzyme predominantly responsible for the normal methemoglobin reduction (NADH-dependent methemoglobin reductase) (9).

Winton reports on a study (10) where methemoglobin levels in blood were measured on infants to determine subclinical effects. He indicates that at intakes over 10 mg of nitrate ion per kilogram of body weight (2.2 mg/kg measured as nitrate nitrogen) the methemoglobin concentration is slightly elevated over normal. The methemoglobin levels returned to normal when the babies were changed to bottled water free of nitrate nitrogen. When a baby is fed a dehydrated formula that is made with water that the mother boils, (increasing the concentration), the intake of 2.2 mg NO_3-N/kilogram can be reached if the water contains 10 mg/l nitrate nitrogen. To determine if a slight elevation of an infant's methemoglobin concentration has an adverse health effect will require a large and elaborate study.

In some circumstances, which are not understood, the standard does not have a safety factor. Cases of illness might occur, but for the usual situation the limit of 10 mg/l NO_3-N will protect the majority of infants.

Older children and adults do not seem to be affected, but the Russian literature reports (11) elevated methemoglobin in school children where water concentrations of NO_3-N were high, 182 mg/l.

Treatment methods to reduce the nitrate content of drinking water are being developed and should be applied when they are ready if another source of water cannot be used. If a water supply cannot maintain the NO_3-N concentration below the limit, diligent efforts must be made to assure that the water is not used for infant feeding. Consumption of water with a high concentration of NO_3-N for as short a period as a day may result in the occurrence of methemoglobinemia.

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ORGANICS-CARBON ADSORBABLE

The possibility of the presence of taste and odor producing substances and toxic organic chemicals in drinking water are of concern to all connected with the provision of safe, esthetically pleasing water to the American consumer. If the quality of drinking water is to be protected, monitoring of organics should be part of any quality control program. Difficulties arise, however, since monitoring for many specific organics is beyond the capabilities of most water supplies at this time (1973). This problem can be overcome somewhat, however, by monitoring for the general organic content of water and assuming, as is done with the total coliform test as the indicator test for pathogens, that if this indicator parameter is below a certain limit, the likelihood of odorous or toxic organics causing problems is reduced.

Historically, the general organic content of drinking water has been determined by measuring the Carbon Chloroform Extract (CCE) and Carbon Alcohol Extract (CAE)(1) concentrations. These extracts have an operational definition and are a mixture of organic compounds that can be absorbed into activated carbon under prescribed conditions and then desorbed with organic solvents under prescribed conditions.

The 1962 Public Health Service Drinking Water Standards contained a limit of 0.2 mg/l for CCE collected with the Carbon Adsorption Method (CAM) sampler (2) operated at a flow-rate of 0.25 gallons (945 ml) per minute, called the high-flow CAM sampler. (Note, because the recovery of organics from water is influenced by the collection and extraction method, lower-case letters are used to distinguish the analytical procedures. Carbon-Chloroform Extracts collected with the high-flow CAM sampler are hereafter called CCE-hf).

Middleton and Rosen (3) detected substituted benzene compounds, kerosene, polycyclic hydrocarbons, phenylether, acrylonitrile and insecticides in various CCE-hf's. This list has been expanded by many investigators in the subsequent years, for example, by Kleopfer and Fairless (4). In 1963, Heuper and Payne (5) reported the carcinogenic properties of finished water CCE-hf's.

In 1965, Booth, English and McDermott (6) developed a CAM sampler similar to the High-Flow CAM Sampler, but with a longer contact time between the sample and the activated carbon. This sampler, called the low-flow CAM sampler, increased organic adsorption and, therefore, overall yield of the determination. In addition, measurement of CAE was included in this method. Extracts from this procedure are called CCE-lf and CAE-lf. No drinking water standard was promulgated for these parameters. Rosen, Mashni, and Safferman (7) isolated odorous organics from a CCE-lf.

Since that time, a CAM sampler, called the "Mini-Sampler," with the advantages of the low-flow CAM sampler, but more reliable, less expensive, smaller, and more convenient, has been developed (8). In addition, the Mini-sampler uses a type of coal-based granular activated carbon that enhances organic collection, thereby increasing the yield of the method. The extraction apparatus has also been minaturized to be more convenient and less expensive and the procedure has been modified to be more vigorous, thereby increasing desorption and further increasing organic recovery (10). The extract from this procedure is called CCE-m.

Tardiff and Deinzer (9) tested the toxicity of a CCE-m collected from the finished water of a river supply. The resulting LD50 of 32 mg/kg would classify this extract as extremely toxic on a typical toxicological scale.

Symons, Love, Buelow and Robeck (10) reported the identification of ε-Caprolactam and 2-Hydroxyadiponitrile by gas chromatography and mass spectrometry in a CCE-m collected from a finished water from a different river supply. This indicates the presence of synthetic organics in this extract.

Extraction with the less polar solvent chloroform does not desorb all of the organics adsorbed onto the activated carbon. Extraction with other solvents has been proposed as a method of monitoring these materials. The use of the polar solvent 95% ethyl alcohol does extract different organics, but it also recovers inorganic salts that were adsorbed on the activated carbon. At this time no reliable technique has been developed for measuring these other organics.

In an effort to determine the range of CCE-m concentrations in finished water, as was done by Ettinger (11), for raw water and Taylor (12), the Interstate Carrier Surveillance Program (13) and the 1969 Community Water Supply Survey (14) for finished water, using the high-flow CAM technique, studies were made with the Mini-sampler at 128 locations. These were all surface water sources, and had varying histories of raw water contamination by organics and taste and odor problems. These sources were in 31 states, the District of Columbia, and Puerto Rico. Single samples were collected at 122 locations and from 2 to 34 samples at the other six locations. These latter data were averaged.

The data were pooled and grouped by extract concentration and the percentage in each concentration category calculated. From these data the percentage of locations with CCE-m concentrations greater than a given concentration was calculated. These are shown in Table II.

The proposed use of a CCE maximum contaminant level was an attempt to deal with gross organic pollution as soon as possible pending the results of further research, and surveys that are planned by EPA and of the NAS study that is required in the Safe Drinking Water Act. CCE was initially used as a means of taste and odor control. As concern over adverse health effects of organic chemicals grew, CCE was turned to as a rough surrogate for organics to be used as a health-based standard rather than as an esthetic standard. Unfortunately,

TABLE II

CCE-m Concentration mg/l	% of Locations* with Concentration Greater Than Given Concentration
0.0	100.0
0.1	97.7
0.2	86.8
0.3	63.4
0.4	39.2
0.5	24.4
0.6	14.2
0.7	7.9
0.8	5.6
0.9	4.0
1.0	3.2
1.1	2.4
1.4	1.6
1.5	0.8
2.3	0.0

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*Based on 128 locations.

as more is learned about organic chemical pollution of drinking water, CCE looks less and less effective as a surrogate for harmful organics.

The principal difficulty with CCE is that it includes only about one-fifth of the total organic content of the volume of water sampled, and it does not measure organic compounds of greatest concern, such as the volatile halomethanes. Thus, a high CCE test result does not necessarily mean that the water tested may pose a hazard to health, and a low CCE test result may be obtained from water with a high level of potentially harmful organic compounds. In short, there is no sound basis of correlation between CCE test results and the level of harmful organic chemicals in the water tested.

To establish a maximum contaminant level under these circumstances would almost certainly do more harm than good. It could give a false sense of security to persons served by systems which are within the established level and a false sense of alarm to persons served by systems which exceed the level. It also would divert resources and attention from efforts to find more effective ways of dealing with the organic chemical problem.

Total organic carbon (TOC) and chemical oxygen demand (COD) are surrogates that have been considered, but they have limitations also. TOC has the advantage of being quicker and cheaper (on a per sample basis) than CCE, but the availability of sensitive instruments for this measurement is questionable. More investigation of the significance of any TOC number as a health effects limit is also needed. COD is easily determined with readily available laboratory equipment, but COD is not limited to organic compounds, and besides a COD number also cannot be adequately related to health significance at this time.

EPA is diverting substantial resources to research into the health effects of specific organic chemicals and groups of organic chemicals. Also, it is expected that the study of the National Academy of Sciences will produce further data on health effects. However, in view of the significance of the potential health problem, it is not enough to wait for this additional health effects data. EPA therefore will undertake to identify one or more surrogate tests for organic chemicals or organic chemical groups, and will also study in depth the presence of specific organic chemicals in drinking water supplies. It is anticipated that this effort will result in the development of an additional MCL or MCL's for organic chemicals by amendment of the Interim Primary Drinking Water Regulations without having to wait for a more complete resolution of the organic chemicals question in the Revised Regulations.

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PESTICIDES

A. Chlorinated Hydrocarbon Insecticides

The chlorinated hydrocarbons are one of the most important groups of synthetic organic insecticides because of their wide use, great stability in the environment, and toxicity to mammals and insects. When absorbed into the body, some of the chlorinated hydrocarbons are not metabolized rapidly but are stored in the fat.

As a general group of insecticides, the chlorinated hydrocarbons can be absorbed into the body through the lungs, the gastro-intestinal tract, or the skin. The symptoms of poisoning, regardless of the compound involved or the route of entry, are similar but may vary in severity. Mild cases of poisoning are characterized by headache, dizziness, gastro-intestinal disturbances, numbness and weakness of the extremities, apprehension, and hyperirritability. In severe cases, there are muscular fasciculations spreading from the head to the extremities, followed eventually by spasms involving whole muscle groups, leading finally to convulsions and death from cardiac or respiratory arrest. The severity of symptoms is related to the concentration of the insecticides in the nervous system, primarily the brain (1).

Criteria Based on Chronic Toxicity

Except as noted below, the approval limits (AL's) for chlorinated hydrocarbons in drinking water have been calculated primarily on the basis of the extrapolated human intake that would be equivalent to that causing minimal toxic effects in mammals (rats and dogs). Table I lists the levels of several chlorinated hydrocarbons fed chronically to dogs and rats (2, 3, 4) that produced minimal toxicity or no effects.

For comparison, the dietary levels are converted to mg/kg body weight/day. Endrin and lindane had lower minimal effect/no-effect levels in dogs than in rats; whereas, for toxaphene and methoxychlor the converse was observed.

Human studies have also been conducted for methoxychlor, although they were of short duration (8 weeks). The highest level tested for methoxychlor was 2 mg/kg/day (5). No illness was reported in these subjects.

Such data from human and animal investigations may be used to derive exposure standards, as for drinking water, by adjusting for factors that influence toxicity such as inter- and intra- species variability, length of exposure, and extensiveness of the studies. To determine a "safe" exposure level for man, conventionally a factor of 1/10 is applied to the data derived from human exposure studies conducted longer than 2 months at which no effects have been observed; whereas, a factor of 1/100 is applied to data derived from human exposure studies conducted for 2 months or less as is the case for the human methoxychlor data cited. A 1/100 factor is applied to animal data when adequate human data are available for corroboration and a factor of 1/500 is generally used on animal data when no adequate and comparable human data are available. The minimal effect levels of endrin, lindane, and toxaphene are adjusted by 1/500 since no adequate data are available for comparison. These derived values are considered the maximum safe exposure levels from all sources. Since these values are expressed as mg/kg/day, they are then readjusted for body weight to determine the total quantity to which persons may be safely exposed.

Analysis of the maximum safe levels (mg/man/day) reveals that these levels are not exactly the same when one species is compared with another. The choice of a level on which to base an AL for water requires the selection of the lowest value from animal experimentation, provided that the human data are within the same order of magnitude. Thus the human data should substantiate the fact that man is no more sensitive to a particular agent than is the rat or the dog.

To set a standard for a particular medium necessitates that account be taken for exposure from other media. In case of the chlorinated hydrocarbons, exposure is expected to occur mostly through the diet. Occasionally, aerial sprays of these agents will result in their inhalation. Dietary intake of pesticide chemicals has been determined by the investigations of the Food and Drug Administration from "market basket" samples of food and water. Duggan and Corneliussen (6) report on this activity from 1964-1970. The average dietary intakes (mg/man/day) are listed in Table I. Comparing the intake from the diet with what are considered acceptable safe levels of these pesticides, it is apparent that only traces of methoxychlor and toxaphene are present in the diet. Less than 10% of the maximum safe level of endrin or lindane are ingested with the diet.

The AL's for chlorinated hydrocarbon insecticides reflect only a portion of man's total exposure to the compounds. In general, 20% of the total acceptable intake is taken to be a reasonable apportionment to water. However, the AL for toxaphene was lowered because of organoleptic effects (7, 8) at concentrations above 0.005 mg/l.

The approval limits for the chlorinated hydrocarbon insecticides are listed in Table I. These limits are meant to serve only in the event that these chemicals are inadvertently present in the water. Deliberate addition of these compounds is neither implied nor sanctioned.

TABLE I. DERIVATION OF APPROVAL LIMITS (AL's) FOR CHLORINATED HYDROCARBON INSECTICIDES

		Lowest Long-Term Levels With Minimal or no Effects		Calculated Maximum Safe Levels			Intake from Diet		Water	
Compound	Species	ppm in diet	mg/kg body weight/day[a]	Safety Factor (X)	mg/kg/day	mg/man/day[b]	mg/man/day (6)	% of Safe Level	% of Safe Level	Recommended MAL (mg/l)[c]
Endrin	Rat Dog Man	5.0(3) 1.0(3) N.A.	0.83 0.02 N.A.	1/500 1/500 -	0.00166 0.00004 -	0.1162 0.0026[d] -	0.00035	4.1	20	0.0002
Lindane	Rat Dog Man	50.0(2) 15.0(2) N.A.	8.3 0.3 N.A.	1/500 1/500 -	0.0166 0.0006 -	1.162 0.042[d] -	0.0035	8.3	20	0.004
Methoxychlor	Rat Dog Man	100.0(2) 4000.0(2) -	17.0 80.0 2.0(5)	1/100 1/100 1/100	0.17 0.3 0.02	11.9 21.0 1.4[d]	T	T	20	0.1
Toxaphene	Rat Dog Man	10.0(2) 400.0(2) N.A.	1.7 8.0 N.A.	1/500 1/500 -	0.0034 0.016 -	0.238[d] 1.12 -	T	T	20	0.005[e] (0.025)[f]

[a] Assume weight of rat = 0.3 kg and of dog = 10 kg; assume average daily food consumption of rat = 0.05 kg and of dog = 0.2 kg.

[b] Assume average weight of human adult = 70 kg.

[c] Assume average daily intake of water for man = 2 liters.

[d] Chosen as basis on which to derive MAL.

[e] Adjusted for organoleptic effects.

[f] Calculated MAL in parentheses.

NA - no data available.

T - infrequent occurrence in trace quantities.

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Criteria Based on Potential Carcinogenicity

To establish AL's for DDT, aldrin, and dieldrin, a different method for deriving AL's must be used, since there is evidence that DDT, aldrin, and dieldrin represent a potential carcinogenic hazard to humans, based on experiments with rats and mice (9, 10, 11, 12). Aldrin is readily converted to dieldrin by animals, soil microorganisms, and insects, and thus the potential carcinogenicity of aldrin will be considered to be equivalent to that of dieldrin (13).

It is recognized that scientists have yet to determine if there is any level of exposure to chemical carcinogens that is completely free of risk of cancer. For the purpose of setting these standards we will assume that the risk of inducing cancer decreases with decreasing dose. Thus, the limits for these possible carcinogens will be derived by estimating the health risk associated with various concentrations and comparing these concentrations with ambient levels to assess the attainability of the proposed limits with presently known means of technology.

Monitoring data available from the Community Water Supply Studies Program (CWSS) carried out during 1969 to 1971 are too questionable to be used as a basis for any conclusions. Original records of the analyses were lost during Hurricane Camille. The only other record which might indicate the ambient level of chlorinated pesticides in drinking water supplies is a survey by the Federal Water Pollution Control Administration published in 1969 as "Pesticides in Surface Waters of the United States -- A Five Year Summary (1964-1968)". Obviously one cannot make the assumption that all of the surface waters analyzed in this survey were utilized as drinking water supplies, and no definite conclusions can be reached on ambient levels in drinking water based on these data.

Since so little information is available concerning the concentrations of aldrin, dieldrin and DDT currently in the nation's drinking waters, EPA has decided to delay the proposal of limits for these compounds pending the completion of a survey of selected water supplies to estimate the extent of current pesticide levels in U. S. drinking water supplies. This survey should be completed within six months.

Upon the completion of this survey, limits for aldrin, dieldrin and DDT will be proposed, based upon an analysis of the health risks associated with low levels of intake of these pesticides and available information concerning attainability. Risk estimates at very low levels of exposure are subject to great uncertainties, but the best available methods for making such estimates will be used. Extrapolation techniques such as the "one-hit" model and the Mantel-Bryan use of the probit model (14) are being intensively reviewed by several agencies of the federal government. Every effort will be made to set the limits for these pesticides at concentrations which will adequately protect public health without imposing economic hardship. The Agency believes that limits far more stringent than those considered in the past should be promulgated.

Aldrin-Dieldrin

Experiments carried out on mice (strain CFl) fed dieldrin in their daily diet, at levels varying from 0.1 to 20 ppm during their normal life span, resulted in significant increases in the incidence of liver tumors (ll). The results of this study appear to be, at present, the most appropriate for calculating the risk associated with a range of concentrations of dieldrin in drinking water.

DDT:

Although earlier studies of the carcinogenic effect of DDT have yielded generally negative results, three recent studies in experimental animals conflict with these previous findings. Using tumor-susceptible hybrid strains of mice, Innes et al (15) produced significantly increased incidences of tumors with the administration of large doses of DDT (46.4 mg/kg/day). In a separate study in mice extending over five generations, a dietary level of 3 ppm of DDT produced a greater incidence of leukemia and malignancies beginning with the F2 and F3 generations (16).

More recent information (12) on the effect of DDT on long-term exposure in mice indicated a higher incidence of liver tumors in the treated population. CF-1 minimal inbred mice were given technical DDT mixed into the diet at the dose levels at 2, 10, 50 and 250 parts per million (ppm) for the entire life span for two consecutive generations. Exposure to all four levels of DDT resulted in a significant increase of liver tumors in males, this being most evident at the highest level used. In females, the incidence of liver tumors was slightly increased following exposure to 250 ppm. In DDT-treated animals the liver tumors were observed at an earlier age than in untreated controls. The age at death with liver tumors and the incidence of liver tumors appear to be directly related to the dose of DDT to which the mice were exposed. Four liver tumors, all occurring in DDT-treated mice, gave metastases. Histologically, liver tumors were either well-differentiated nodular growths, pressing but not infiltrating the surrounding parenchyma, or nodular growths in which the architecture of the liver was obliterated showing glandular or trabecular patterns. The results of this study appear to be, at present, the most appropriate to use as a basis for extrapolating the risk associated with a range of concentrations of DDT in drinking water.

Chlordane and Heptachlor

Because recent evidence also implicates chlordane and heptachlor as potential carcinogens, establishment of limits for these pesticides must be based on considerations similar to those for aldrin, dieldrin and DDT.

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B. Chlorophenoxy Herbicides

Aquatic weeds have become substantial problems in the U.S. in recent years, and chemical control of this vegetation has won wide acceptance. Since waters to which applications of herbicides are made are sometimes employed as raw water sources of drinking water, there is the possibility that herbicides may enter potable source water. Consequently, a standard is needed for the more extensively used herbicides so as to protect the health of the water consumer.

Two widely used herbicides are 2,4-D (2,4-dichlorophenoxyacetic acid) and 2,4,5-TP (silvex) [2-(2,4,5-trichlorophenoxy) propionic acid]. [A closely related compound, 2,4,5-T (2,4,5-trichlorophenoxyacetic acid) had been extensively used at one time, but has been banned for major aquatic uses.] Each of these compounds is formulated in a variety of salts and esters that may have a marked difference in herbicidal properties, but all of which are hydrolyzed rapidly to the corresponding acid in the body.

The acute toxicity following oral administration to a number of experimental animals is moderate. Studies (1-4) of the acute oral toxicity of the chlorinated phenoxyalkyl acids indicate that there is approximately a three-fold variation between the species of animals studied. It appears that acute oral toxicity of the three compounds is of about the same magnitude within each species (e.g., in the rat, an oral LD of about 500 mg/kg for each agent).

The subacute oral toxicity of chlorophenoxy herbicides has been investigated in a number of species of experimental animals (1-6). The dog was the most sensitive species studied and often displayed mild injury in response to doses of 10 mg/kg/day for 90 days, and serious effects from a dose of 20 mg/kg/day for 90 days. Lehman (6) reported that the no-effect level of 2, 4-D is 50* mg/kg/day in the rat, and 8.0 mg/kg/day in the dog.

Although 2, 4, 5-T has been banned for all aquatic uses there is considerable interest as to why this action was taken, so for informational purposes, a discussion of the toxicity of this herbicide is included. In a study of various pesticides and related compounds for teratogenic effects, Cortney, et al. (7) noted terata and embryotoxicity from 2, 4, 5-T. These effects were evidenced by statistically increased proportions of litters affected and of abnormal fetuses within the litters (notably, cleft palate and cystic kidneys). Effects were noted in both mice and rats, although the rat appeared to be more sensitive to this effect. A dosage of 21.5 mg/kg produced no harmful effects in mice, while a level of 4.6 mg/kg caused minimal, but statistically significant, effects in the rat. More recent work (8) has indicated that a contaminant (2, 3, 7, 8-tetrachlorodibenzo-p-dioxin) which was present at approximately 30 ppm in the 2, 4, 5-T formulation originally tested was highly toxic to experimental animals and produced fetal and maternal toxicity at levels as low as 0.0005 mg/kg. However, purified 2, 4, 5-T has also produced teratogenic effects in both hamsters and rats at relatively high dosage rates (9). Current production samples of 2, 4, 5-T that contain less than 1 ppm of dioxin did not produce embryotoxicity or terata in rats at levels as high as 24 mg/kg/day (10).

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*In the March 14, 1975, issue of this document, this figure was erroneously written as 0.5.

The subacute and chronic toxicity of 2, 4, 5-TP has been studied in experimental animals (11). The results of 90-day feeding studies indicate that the no-effect levels of the sodium and potassium salts of 2, 4, 5-TP are 2 mg/kg/day in rats, and 13 mg/kg/day in dogs. In 2-year feeding studies with these same salts, the no-effect levels were 2.6 mg/kg/day in rats and 0.9 mg/kg/day in dogs.

Some data are available on the toxicity of 2, 4-D to man. A daily dosage of 500 mg (about 7 mg/kg) produced no apparent ill effects in a volunteer over a 21-day period (12). When 2, 4-D was investigated as a possible treatment for disseminated coccidioidomycosis, the patient had no side effects from 18 intravenous doses during 33 days; each of the last 12 doses in the series was 800 mg (about 15 mg/kg) or more, the last being 2000 mg (about 37 mg/kg) (13). A nineteenth and final dose of 3600 mg (67 mg/kg) produced mild symptoms.

The acute oral dose of 2, 4-D required to produce symptoms in man is probably 3000 to 4000 mg (or about 45 to 60 mg/kg). A comparison of other toxicity values for 2, 4, 5-TP indicates that the toxicity of these two agents is of the same order of magnitude. Thus, in the absence of any specific toxicologic data for 2, 4, 5-TP in man, it might be estimated that the acute oral dose of 2, 4, 5-TP required to produce symptoms in man would also be about 3000 to 4000 mg.

In addition to these specific data, the favorable record of use experience of 2,4-D is also pertinent. Sixty-three million pounds of 2,4-D were produced in 1965 while there were no confirmed cases of occupational poisoning and few instances of any illness due to ingestions (14, 15). One case of 2,4-D poisoning in man has been reported by Berwick (16).

Table I displays the derivation of the approval limits for the two chlorophenoxy herbicides most widely used. The long-term no-effect levels (mg/kg/day) are listed for the rat and the dog. These values are adjusted by 1/500 for 2,4-D and 2.4.5-TP. The safe levels are then readjusted to reflect total allowable intake per person. Since little 2,4-D or 2,4,5-TP are expected to occur in foods, 20% of the safe exposure level can be reasonably allocated to water without jeopardizing the health of the consumer.

The approval limits for these herbicides are meant to serve in the event that these chemicals inadvertently occur in the water. Deliberate addition of these compounds to drinking water sources is neither implied nor sanctioned.

TABLE I. DERIVATION OF APPROVAL LIMITS (AL) FOR CHLOROPHENOXY HERBICIDES

Compound	Lowest Long-Term Levels with Minimal or No Effects		Calculated Maximum Safe Levels From all Sources of Exposure			Water	
	Species	mg/kg/day[a]	Safety Factor (X)	mg/kg/day	mg/man/day[b]	% of Safe Level	AL (mg/l)[c]
2,4-D	Rat	50 (6)	1/500	0.1	7.0	20	0.1
	Dog	8.0 (6)	1/500	0.016	1.12[d]		
2,4,5-TP	Rat	2.6 (12)	1/500	0.005	0.35	20	0.01
	Dog	0.9 (12)	1/500	0.002	0.14[d]		

[a] Assume weight of rat = 0.3 kg and of dog = 10 kg; assume average daily food consumption of rat = 0.05 kg and of dog = 0.2 kg.

[b] Assume average weight of human adult = 70 kg.

[c] Assume average daily intake of water for man = 2 liters.

[d] Chosen as basis on which to derive AL.

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SELENIUM

The 1962 Drinking Water Standards Committee lowered the limit for selenium in drinking water primarily out of concern over the possible carcinogenic properties of the element. Data supporting the carcinogenicity of selenium has not been forthcoming, and more recent findings concerning the nutritional requirement for selenium has required a comprehensive review of the data available concerning the toxicity of selenium and its compounds.

The controversy over the present limits of selenium acceptable in the environment is largely the result of the demonstration by Schwarz and Foltz (2) that the element was an integral part of "factor 3," recognized for some time as essential in animal nutrition. While definite evidence is still lacking for a nutritional requirement for selenium in man, certain cases of protein-resistant kwashiorkor have been shown to be responsive to administration of the element (3).

Consideration of a maximal concentration of selenium allowable in drinking water is further complicated by the many secondary factors known to affect both the efficacy of selenium in alleviating deficiency syndromes and the intakes associated with toxicity. The chemical form of selenium (4), the protein content of the diet (5), the source of dietary protein (6), the presence of other trace elements (1, 7, 8), and the vitamin E intake (9, 10, 11) all affect the beneficial and/or adverse effects of selenium in experimental animals. The fact that these interactions are not simple is illustrated by the comments of Frost (1) on the well-known antagonism of arsenic in selenium toxicity (1, 7, 8, 12). He has found that arsenic in drinking water accentuates the toxicity of selenium in drinking water in contrast to the protective effect of arsenic seen when selenium was administered via the diet. Consequently, when considering "safe" levels of selenium in drinking water, consideration must also be given to the variability in these other factors which are certain to occur in any given population.

The current limit of 0.01 mg/liter of selenium in drinking water is based on the total selenium content. No systematic investigation of the forms of selenium in drinking water sources with excessive concentrations has ever been carried out. Since elemental selenium must be oxidized to selenite or selenate before it has appreciable solubility in water (13), one would predict that these would be the principal inorganic forms that occur in water. Organic forms of selenium occur in seleniferous soils and have sufficient mobility in an aqueous environment to be preferentially absorbed over selenate in certain plants (14). However, the extent to which these compounds might occur in source waters is essentially unknown.

There is considerable difficulty involved in determining what the required level and toxic levels of selenium intake in humans might be. The basic problem is that dietary selenium includes an unknown variety of selenium compounds in varying mixtures. Toxicologic examination of plant sources of selenium has revealed that selenium present in seleniferous grains is more toxic than inorganic selenium added to the diet (16). Although there is a fairly extensive literature on industrial exposures to selenium (see Cerwenka and Cooper, 1961 (17), and Cooper, 1967 (18) for reviews of this subject), the results do not apply well to environmental exposures since the only studies that made an attempt to document systemic absorption involved elemental selenium (19). Elemental selenium is virtually non-toxic to plants and animals that have been shown to be very sensitive to the water soluble forms of selenium.

Only one documented case of human selenium toxicity for a water source uncomplicated with selenium in the diet has been reported (21). Members of an Indian family developed loss of hair, weakened nails, and listlessness after only 3 months' exposure to well-water containing 9 mg/l. The children in the family showed increased mental alertness after use of water from the seleniferous well was discontinued, as evidenced by better work in school (22).

Smith and co-workers (23, 24) reported the results of their studies dealing with human exposure to high environmental selenium concentrations in the 1930's. They reported a high incidence of gastrointestinal problems, bad teeth, and an icteriod skin color in seleniferous areas. The individuals exhibiting these symptoms had urinary selenium levels of 0.2-1.98 µg/liter as compared to the 0.0-0.15 µg/liter that Glover (19) indicates to be the normal range. The gastrointestinal disturbances and the icteriod discoloration of the skin apparently have their counterparts in the anorexia (23) and bilirubinemia (7), respectively, in rats fed selenium. The effect of selenium on teeth has had some marginal documentation in rats (26); and has been supported by Hadjimarkos (27) and refuted by Cadell and Cousins (28) in epidemiologic studies.

From urinary concentrations of selenium, Smith and Westfall (24) estimated that the individuals displaying these symptoms were ingesting 0.01 to 0.10 mg/kg/day, and possibly as much as 0.20 mg/kg/day. For the 70 kg man, this would amount to a daily intake of 700 to 7000 ug/day. Smith (24, 29) also presented the range of selenium concentrations found in various food classes in the areas in which the field studies had been conducted. With the use of the table provided in Dietary Levels of Households in the U.S., Spring 1965 (U.S.D.A. Agri. Res. Service), calculations from these data result in a range of intake of 600-6300 ug/day, very close to the estimates made from urinary concentrations of selenium. These intakes of selenium correspond in the main with the levels producing adverse effects in other mammalian species. Tinsley et al. (25) found that an intake of 0.125 mg/kg/day adversely affected early growth in rats. 1.1 mg/kg, administered twice weekly (ca. 0.3 mg/kg/day), has been found to adversely affect growth and to increase mortality in Hereford steers (30). Mortality in ewes was increased at 0.825 mg/kg/day. The steers were administered sodium selenite; the ewes sodium selenate. Although these levels are slightly higher than those reported for the human exposures, it must be remembered that the parameters measured would not be acceptable either in terms of severity or incidence in the human population.

Few studies have been performed to specifically examine the toxicity of selenium administered in drinking water. Pletnikova (31) found the rabbit to be very sensitive to selenium as selenite. Ten µg/l in drinking water resulted in a 40% reduction in the elimination of bromosulphalein by the liver. Since no apparent consideration was given to the selenium content of the diet of these animals, the meaning of this result in terms of liver function is obscure. If the sole intake of selenium were from the water in these studies, the controls had to be deficient and the experimental group marginal, at best, in terms of the dietary requirement for selenium. The duration of the study was 7 1/2 months. Schroeder (32) has indicated that intake of selenite from drinking water is more toxic than when mixed with food. However, this suggestion was not based on a direct experimental comparison. Rosenfeld and Beath (33) studied the effects of sodium selenate in drinking water on reproduction in rats. Selenium concentrations of 2.5 mg/l reduced the number of young reared by the second generation of mothers, and 7.5 mg/l prevented reproduction in females.

Early work (34), using both naturally occurring, and a selenide salt, indicated the formation of adenomas and low-grade non-metastasizing hepatic cell carcinomas in 11 of 53 rats surviving 18 months of diets containing selenium. Harr et al. (24), in a much more extensive study using selenite and selenate salts, found no evidence of neoplasms that could be attributed to the addition of these selenium compounds to the diet at 0.5 - 16 ppm. Volganev and Tschenkes (35) negated their earlier results, which had indicated that 4.3 mg/l selenium as selenite in the diet gave rise to tumors, but had not used proper controls. It should be noted that these studies are not a direct negation of the earlier studies implicating selenium as a carcinogen, since entirely different compounds of selenium were used in the early work. Consequently, the possibility that other compounds of selenium, besides selenite and selenate, possess carcinogenic properties cannot be strictly ruled out. The carcinogenic properties of selenium are further complicated by recent reports of the effectiveness of selenium, 1 mg/l (as selenite), in reducing papillomas induced by various chemicals in mice (36).

Any consideration of a maximum allowable concentration of selenium must include the evidence that the element is an essential dietary requirement. A range of 0.04 to 0.10 mg/l in the diet is considered adequate to protect animals from the various manifestations of selenium deficiency (10, 37, 38). Using the recent data on Morris and Levander (39), an estimate of the present average daily intake of selenium by the American population may be calculated. This figure approximates 200 ug/day and some variation around this figure would be anticipated primarily as the result of individual preferences, particularly in meats. Since no deficiency diseases of selenium have been reported to date in the U.S., it may be assumed that 200 ug/day of selenium is nutritionally adequate.

Signs of selenium toxicity have been seen at an estimated level of selenium intake of 0.7-7 mg/day according to the data of Smith et al. (23, 24). At the present limit on selenium content of drinking water, water would increase the basal 200 ug/day intake of selenium by only 10%, if one assumes a 2-liter ingestion of water per day. This results in a minimum safety factor of 3, considering the lower end of the range of selenium intakes that have been associated with minor toxic effects in man. In view of the relative scarcity of data directly applicable to the apparent small margin of safety brought about by selenium contained in the diet, selenium concentrations above 0.01 mg/liter shall not be permitted in the drinking water.

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SILVER

The need to set a water standard for silver (Ag) arises from its intentional addition to waters as a disinfectant. The chief effect of silver in the body is cosmetic. It consists of a permanent blue-grey discoloration of the skin, eyes, and mucous membranes which is unsightly and disturbing to the observer as well as to the victim. The amount of colloidal silver required to produce this condition (argyria, argyrosis), and to serve as a basis of determining the water standard, in not known, however, but the amount of silver from injected Ag-arphenamine, which produces argyria is precisely known. This value is any amount greater than 1 gram of silver, 8g Ag-arsphenamine, in an adult (1, 2).

From a review (2) of more than 200 cases of argyria, the following additional facts were derived. Most common salts of silver produce argyria when ingested or injected in sufficient doses. There is a long-delayed appearance of discoloration. No case has been uncovered that has resulted from an idiosyncrasy to silver. There was, however, considerable variability in predisposition to argyria; the cause of this is unknown, but individuals concurrently receiving bismuth medication developed argyria more readily. Although there is no evidence that gradual deposition of silver in the body produces any significant alteration in physiologic function, authorities are of the opinion that occasional mild systemic effects from silver may have been overshadowed by the striking external changes. In this connection, there is a report (3) of implanted silver amalgams resulting in localized argyria restricted to the elastic fibers and capillaries. The histopathologic reaction resembed a blue nevus simulating a neoplasm with filamentous structures and globular masses. Silver affinity for elastic fibers had been noted a half-century earlier (5).

A study (5) of the metabolism of silver from intragastric intake in the rat, using radio-silver in carrier-free tracer amounts, showed absorption to be less than 0.1-0.2 percent of the silver administered; but this evidence is inconclusive because of the rapid elimination of silver when given in carrier-free amounts. Further study indicated, however, that silver is primarily excreted by the liver. This would be particularly true if the silver were in colloidal form. Silver in the body is transported chiefly by the blood stream in which the plasma proteins and the red cells carry pratically all of it in extremely labile combinations. The half-time of small amounts of silver in the blood stream of the rat was about 1 hour. A later report (6), using the spectrographic method on normal human blood, showed silver unmistakably in the red blood cell and questionably in the red cell ghosts and in the plasma. Once silver is fixed in the tissues, however, negligible excretion occurs in the urine (7).

A study (8) of the toxicologic effects of silver added to drinking water of rats at concentrations up to 1,000 µg/l (nature of the silver salt unstated) showed pathologic changes in kidneys, liver, and spleen at 400, 700, and 1,000 µg/l, respectively.

A study (9) of the resorption of silver through human skin using radio-silver Ag^{111} has shown none passing the dermal barrier from either solution (2 percent $AgNO_3$) or ointment, within limits of experimental error (± 2 percent). This would indicate no significant addition of silver to the body from bathing waters treated with silver.

Uncertainty currently surrounds any evaluation of the amount of silver introduced into the body when silver-treated water is used for culinary purposes. It is reasonable to assume that vegtables belonging to the family Brassicoceae, such as cabbage, turnips, cauliflower, and onions, would combine with residual silver in the cooking water. The silver content of several liters of water could thus be ingested.

Because of the evidence (7) that silver, once absorbed, is held indefinitely in tissues, particularly the skin, without evident loss through usual channels of elimination or reduction by transmigration to other body sites, and because of the probable high absorbability of silver bound to sulfur components of food cooked in silver-containing waters [the intake for which absorption was reported in 1940 to amount to 60-80 µg per day (10)], the concentration of silver in drinking water shall not exceed 0.05 mg/l.

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SODIUM

Man's intake of sodium is mostly influenced by the use of salt. Intake of sodium chloride for American males is estimated to be 10 grams per day, with a range of 4 to 24 grams (1). This would be a sodium intake of 1600 to 9600 mg per day. Intake of these amounts is considered by most to have no adverse effect on normal individuals. Even Dahl, who has been one of the strong advocates of the need for restricting salt intake, has felt that an intake of 2000 mg of sodium could be allowed for an adult without a family history of hypertension. Intake of sodium from hospital "house" diets has been measured recently (2). The sodium content of a pool of 21 consecutive meals that were seasoned by the chef or the dietitian from twenty selected general hospitals was determined each quarter. The average sodium intake per capita per day was 3625 ± 971 (SD) milligrams. The intake could be greatly changed between individuals who never add salt to the food at the table and the individuals who always add salt even before tasting.

The taste threshold of sodium in water depends on several factors (3). The predominant anion has an effect; the thresholds for sodium were 500 mg/l from sodium chloride, 700 mg/l from sodium nitrate, and 1000 mg/l from sodium sulfate. A heavy salt user had a threshold of taste that was 50 percent higher, and the taste was less detectable in cold water.

Six of 14 infants exposed to a sodium concentration of 21,140 mg/l died when salt was mistakenly used for sugar in their formula (4). Sea water would have about 10,000 mg/l of sodium.

Severe exacerbation of chronic congestive heart failure due to sodium in water has been documented (3). One patient required hospitalization when he changed his source of domestic water to one that had 4200 mg/l sodium. Another patient was readmitted at two-to-three-week intervals when using a source of drinking water of 3500 mg/l sodium.

Sodium-restricted diets are used to control several disease conditions of man. The rationale, complications, and practical aspects of their use were reviewed by a committee on food and nutrition of the National Research Council (5). Sodium-restrictive diets are essential in treating congestive cardiac failure, hypertension, renal disease, cirrhosis of the liver, toxemias of pregnancy, and Meniere's disease.

Hormone therapy with ACTH and cortisone is used for several diseases. Sodium retention is one of the frequent metabolic consequences following administration of these therapeutic agents, and sodium-restricted diets are required, especially for long periods of treatment. More recent medical text books continue to point out the usefulness of sodium-restricted diets for these several diseases where fluid retention is a problem (6).

When disease causes fluid retention in the body, with subsequent edema and ascites, there is a diminished urinary excretion of sodium and of water. If the sodium intake is restricted in these circumstances, further fluid retention will usually not occur, and the excess water ingested will be excreted in the urine because the mechanisms that maintain the concentration of sodium in the extracellular fluid do not permit the retention of water without sodium.

Almost all foods contain some sodium, and it is difficult to provide a nutritionally adequate diet without an intake of about 440 mg of sodium per day from food; this intake would be from the naturally occurring sodium in food with no salt added. The additional 60 mg that would increase the intake to the widely used restricted diet of 500 mg per day must account for all non-nutrition intake that occurs from drugs, water and incidental intakes. A concentration of sodium in drinking water up to 20 mg per liter is considered compatible with this diet. When the sodium content exceeds 20 mg/l, the physician must take this into account to modify the diet or prescribe that distilled water be used. Water utilities that distribute water that exceeds 20 mg/l must inform physicians of the sodium content of the water so that the health of consumers can be protected. About 40 percent of the water supplies are known to exceed 20 mg/l and would be required to keep physicians informed of the sodium concentration (7). Most of the State health departments have made provision for determining the sodium content of drinking water on a routine basis and are now informing physicians in their jurisdiction (8). If change of source or a treatment change such as softening occurs that will significantly increase the sodium concentration, the utility must be sure that all physicians that care for consumers are aware of the impending change. Diets prescribing intakes of less than 500 mg per day must use special foods such as milk with the sodium reduced, or fruits that are naturally low in sodium.

It is not known how many persons are on sodium-restricted diets and to what extent the sodium intake is restricted. To reduce edema or swelling, the physician may prescribe a diuretic drug, a sodium-restricted diet, or a combination of the two. Therapy, of course, depends on the patient's condition, but there are also regional differences that probably result from physician training. The American Heart Association (AHA) (9) feels that diuretics may allow for less need of very restricted diets and that diuretics are necessary for quick results in acute conditions. For long-term use, a sodium-restricted diet is simpler, safer, and more economical for the patient. It is preferable, especially when a moderate or mild sodium-restricted diet will effectively control the patient's hypertension and water retention. Literature is provided to physicians by the AHA to distribute to their patients explaining the sodium-restricted diets. These cover the "strict" restriction - 500 mg sodium, "moderate" restriction - 1000 mg sodium, and the "mild" restricted diet - 2400 to 4500 mg sodium. From 1958 through June 1971, there were 2,365,000 pieces of this literature distributed: 37% - 500 mg; 34% - 1000 mg; and 29% - "mild" (10). There are many ways a physician can counsel his patients other than using this literature, so the total distribution does not reflect the extent of the problem, but the proportion of booklets distributed may provide an estimate of the portion of diets that are prescribed. The "mild" restricted diet could require just cutting down on the use of salt, and literature for the patient would not be as necessary.

The AHA estimates that hypertension affects more the 21 million Americans, and in more than half of these cases put enough strain on the heart to be responsible for the development of hypertensive heart disease (11). Congestive heart failure is a sequelae of several forms of disease that damage the heart and would affect some unknown portion of the 27 million persons with cardiovascular disease. Thus, from 21 to 27 million Americans would be concerned with sodium intake.

Toxemias of pregnancy are common complications of gestation and occur in 6 to 7 percent of all pregnancies in the last trimester (12). Thus, about 230,000 women would be very concerned with sodium intake each year. Other diseases are treated with restricted sodium intake, but no estimate can be made on the number of people involved.

Questions about salt usage were asked on the ninth biennial examination of the National Heart Institute's Framingham, Massachusetts Study (13). The study population was free of coronary heart disease when the study began in 1949 and now are over 45 years of age. There were 3,833 respondents. Forty-five percent of the males and 30 percent of the females reported that they add salt routinely to their food before tasting. But at the other extreme, 9 percent of the men and 14 percent of the women avoid salt intake. More of the people 60 and over avoid salt intake than the 45 to 59 population. It is not determined if the salt restriction was medically prescribed nor how extensively the sodium intake was restricted.

It can be seen that a significant proportion of the population needs to and is trying to curtail its sodium intake. The sodium content of drinking water should not be significantly increased for frivolous reasons. This is particularly true of locations where many of the people using the water would be susceptible to adverse health effects, such as hospitals, nursing homes, and retirement communities. The use of sodium hypochlorite for disinfection, or sodium fluoride for control of tooth decay, would increase the sodium content of drinking water but to an insignificant amount. The use of sodium compounds for corrosion control might cause a significant increase, and softening by either the base exchange or lime-soda ash process would significantly increase the sodium content of drinking water. For each milligram per liter of hardness removed as calcium carbonate by the exchange process, the sodium content would be increased about one-half mg per liter. The increase in excess lime softening would depend on the amount of soda ash added. A study in North Carolina found that the sodium content of 30 private well-water supplies increased from 110 mg/l to 269 mg/l sodium on the average after softening (14). The sodium content of the softened water was much higher shortly after the softener had been regenerated than later in the cycle. A case has been reported where a replacement element type softener was not flushed, and the drinking water had a sodium content of 3, 700 mg/l when the unit was put back in service.

As a further deterrent to softening of water, it should be noted that there is considerable evidence of an inverse relationship between water hardness and certain cardiovascular diseases. Research in the area is being accelerated to determine cause and effect relationships. Until the full significance of water hardness is known, and because of the increase in sodium content of softened waters, utilities should carefuly consider the consequences of installing softening treatment.

All consumers could use the water for drinking if the sodium content was kept below 20 mg per liter, but about 40 percent of the U.S. water supplies have a natural or added sodium content above this concentration (7). Many industrial wastes and runoff from deiced highways may increase the sodium pollution of surface water (15). The problem is most acute when ground water is polluted with sodium (16, 17) because it remains for a long time. Removal of sodium from water requires processes being developed by the Office of Saline Water (18) and are economically feasible only in certain situations.

The person who is required to maintain a restricted sodium intake below 500 mg per day can use a water supply that contains 20 mg or less sodium per liter. If the water supply contains more sodium, low sodium bottled water or specially treated water will have to be used. In the moderately restricted diet that allows for a consumption of 1000 mg sodium per day the food intake is essentially the same, but the diet is liberalized to allow the use of 1/4 teaspoon of salt, some regular bakery bread, and/or some salted butter. If persons on the moderately restricted diet found it necessary to use a water with a significant sodium content they could still maintain their limited sodium intake with a water containing 270 mg/liter. This would require allocating all the liberalized intake to water (the original 20 mg/l and 250 mg/l more with two liter domestic use, drinking or cooking, per day). High sodium in water causes some transfer of sodium to foods cooked in such water (5).

It is essential that the sodium content of public water supplies be known and this information be disseminated to physicians who have patients in the service area. Thus, diets for those who must restrict their sodium intake can be designed to allow for the sodium intake from the public water supply or the persons can be advised to use other sources of drinking water. Special efforts of public notification must be made for supplies that have very high sodium content so that persons on the more restricted sodium intakes will not be overly stressed if they occasionally use these water supplies.

The 1963 Sodium Survey (7) had the following percent distribution of sodium concentration from 2100 public water supplies:

Range of Sodium Ion Concentration	Percent of Total Samples
mg/l	%
0 - 19.9	58.2
20 - 49.9	19.0
50 - 99.9	9.3
100 - 249.9	8.7
250 -399.9	3.6
400 -499.9	0.5
500 -999.9	0.7
Over 1000	0.1

While the question of a maximum contaminant level for sodium is still under consideration by the National Academy of Sciences and others, no specific level will be proposed for the Interim Primary Drinking Water Regulations. The Environmental Protection Agency believes that the available data do not support any particular level for sodium in drinking water, and that the regulation of sodium by a maximum contaminant level is a relatively inflexible, very expensive means of dealing with a problem which varies greatly from person to person.

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SULFATE

The presence of sulfate ion in drinking water can result in a cathartic effect. Both sodium sulfate and magnesium sulfate are well-known laxatives. The laxative dose for both Glauber salt ($Na_2SO_4 \cdot 10H_2O$) and Epsom salt ($MgSO_4 \cdot 7H_2O$) is about two grams. Two liters of water with about 300 mg/l of sulfate derived from Glauber salt, or 390 mg/l of sulfate from Epsom salt, would provide this dose. Calcium sulfate is much less active in this respect.

This laxative effect is commonly noted by newcomers and casual users of waters high in sulfates. One evidently becomes acclimated to use of these waters in a relatively short time.

The North Dakota State Department of Health has collected information on the laxative effects of water as related to mineral quality. This has been obtained by having individuals submitting water samples for mineral analysis complete a questionnaire that asks about the taste and odor of the water, its laxative effect (particularly on those not accustomed to using it), its effect on coffee, and its effect on potatoes cooked in it.

Peterson (1) and Moore (2) have analyzed part of the data collected, particularly with regard to the laxative effect of the water.

Peterson found that, in general, the waters containing more than 750 mg/l of sulfate showed a laxative effect and those with less than 600 mg/l generally did not. If the water was high in magnesium, the effect was shown at lower sulfate concentrations than if other cations were dominant. Moore showed that laxative effects were experienced by the most sensitive persons, not accustomed to the water, when magnesium was about 200 mg/l and by the average person when magnesium was 500-1,000 mg/l. Moore analyzed the data as shown in Table 1. When sulfates plus magnesium exceed 1,000 mg/l, a majority of those who gave a definite reply indicated a laxative effect.

Table 2 presents some data collected by Lockhart, Tucker and Merritt (3) and Whipple (4) on the influence of sulfate on the taste of water and coffee. Because of the milder taste of sulfate over chloride (5)(6) a taste standard for sulfate would probably be in the 300-400 mg/l range. The Peterson data (1) and Table 1 (2), however, indicate that from 600 to 1000 mg/l of sulfate has a laxative effect on a majority of users.

While a limit for sulfate may be included in Secondary Drinking Water Regulations, on the basis of the effect of sulfate on water taste, no maximum contaminant level is being proposed at this time. As noted above, a relatively high concentration of sulfate in drinking water has little or no known effect on regular users of the water, but transients using high sulfate water sometimes experience a laxative effect. Whether this effect will occur, and its severity, varies greatly with such factors as the level of sulfate in the water being consumed and the level of sulfate to which the transient is accustomed. Because of this great variability, the available data do not support the establishment of any given maximum contaminant level. The Environmental Protection Agency recommends that the States institute monitoring programs for sulfates, and that the transients be notified if the sulfate content of the water is high. Such notification should include an assessment of the possible physiological effects of consumption of the water.

In the meantime, research is being undertaken to determine if the health effects of sulfate in drinking water warrant further consideration. If data are generated to support a maximum contaminant level, this level will be proposed for inclusion in Revised Interim Primary Water Regulations.

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